BRAIN:

A JOURNAL OF NEUROLOGY

LIBRARY MEDICAL SCHOOL
UNIVERSITY OF OREGON, PORTLAND

EDITED

FOR THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM

BY

HENRY HEAD, M.D., F.R.S.

Assisted in the selection of Papers by

SIR JOHN BATTY TUKE, M.D., LL.D.

H. C. BASTIAN, M.D., F.R.S.
JUDSON S. BURY, M.D.
W. D. HALLIBURTON, M.D., F.R.S.
SIR VICTOR HORSLEY, F.R.S.
C. A. MERCIER, M.B.
W. JULIUS MICKLE, M.D.
J. S. RISIEN RUSSELL, M.D.
ERNEST H. STARLING, M.D., F.R.S.

VOL. XXVIII.

London:
MACMILLAN AND CO., LIMITED
ST. MARTIN'S STREET, W.C.
NEW YORK: THE MACMILLAN COMPANY
1905

LONDON
PRINTED BY JOHN BALE, SONS & DANIELSSON, LTD.
83-91, GREAT TITCHFIELD STREET, W.

BRAIN.

PART I., 1905.

Original Articles and Clinical Cases.

THE RELATION OF THE LUNACY LAWS TO THE TREATMENT OF INSANITY.

BY SIR JOHN BATTY TUKE, M.D., LL.D., M.P.

Presidential Address Delivered before the Neurological Society of the United Kingdom on February 16, 1905.

The object of this address is to present to the Society as succinctly as possible the relation of the law to the treatment of the insanities, and the disabilities under which we, as a profession, lie in this respect. I feel no apology is needed to the Neurological Society of Great Britain and Ireland for undertaking this task, for I am never tired of quoting the words of Griesinger uttered nearly forty years ago, when he said, "Diseases of the nervous system form one inseparable whole of which the so-called mental diseases only embrace a certain moderate proportion." And I am further supported by the feeling that the vast additions to our knowledge of the structure and function of the nervous system, contributed by members of this Society, have no small bearing on the political position.

I am aware the subject has been under the consideration of certain of our members in one of its aspects, but I propose to regard it from the broadest standpoint, and deal with it

20-174

as it affects the physician, the patient, and the public. It is a question which has occupied my thoughts for many years. I have written about it and spoken about it, but I cannot resist the temptation to avail myself of this exceptional opportunity to reiterate my views, notwithstanding that I shall be subject to the charge of repetition. The questions involved are of such national importance, that I must bow to the criticism if it be advanced.

I believe I am justified in stating that there is a widespread desire for improvements and modifications of the existing Lunacy Laws. This feeling holds with the medical profession for professional reasons, with the public on account of fiscal considerations. We have to deal mainly with the first set of reasons, although in order to buttress them, in order to make them efficacious, we may have to collate them with the second.

My primary contention in support of the demand for reform is that an important and steadily increasing change has come over our conception of so-called mental disease. It would be futile on the present occasion to review in support of this contention the history of the treatment of the insane during the barbaric periods prior to 1800; but it must be remembered that the dregs of the revelations brought to light by various inquiries instituted about that time still exercise some influence on public opinion. The memory of old scandals dies hard. During what may be termed the humanitarian period which may be roughly computed as existing during the first fifty or sixty years of the last century, the study of insanity occupied the attention of many physicians. For the most part, however, their study proceeded on psychological lines, and the prominence of the mental symptoms tended to blur the main issue. It cannot be denied, in fact, it should be proudly asserted, that during this period eminent clinicians arose, of whom Calmeil may be held as the type and the chief, who demonstrated the association of mental symptoms with certain bodily conditions, and who established clinical groups, certain of which still hold ground. Yet proof of the hold maintained by the psychological conception on the medical mind is afforded by the

fact that in the "Nomenclature of Diseases" prepared by the Royal College of Physicians of London, and issued by the Registrar General in 1863, all the insanities were comprised under *six* "Disorders of the Intellect." That such should be the trend of professional and public opinion was inevitable in the then existing state of knowledge.

I have no hesitation in expressing the opinion that during the last forty years a vast change has come over the views of the profession in respect of the insanities, and that that change is due to our extended knowledge of the structure and function of the nervous system acquired during that period, and to the connotation of such knowledge with the advances in general medicine. I am not going to labour this argument by a recapitulation of the various acquisitions of science. I would rather put it this way—I would ask those who are younger amongst us to try to realise what their mental attitude towards such a class of symptoms as characterise the insanities would have been had they lived at a time when the naked-eye topography of the convolutions was undescribed, when nerve tracts were for the most part undifferentiated, when localisation of function of cortical areas was not evolved, when only some two or three men in Great Britain could demonstrate a nerve-cell, and that imperfectly, when the microscope could not be brought to bear on the morbid conditions of the nervous tissues, when, in fact, the very institutes of medicine on which the physician now relies when forming his estimate of the nature of a case were entirely wanting. I do not know what the student of abstract philosophy may think, but I hold that, along with the elaboration of an apparatus, along with the demonstration of the paths of functional activity, a higher general conception of the nature of morbid mental symptoms has been arrived at than was possible when all we had to depend on was vague induction. It would be a work of supererogation, even if time permitted, to follow out this thesis in detail, for I hope I carry the general sense of the meeting with me.

But I desire to indicate what I consider to be the point on which our acquisitions concentrate themselves in respect

of the insanities. We relate in our minds the existence of a delicate and complex apparatus, with definite proof of its susceptibility to the rapid action of disease and with distinct evidence of its power of regeneration. The theory of the dissociation of neuron groups is not merely an inference applied as an explanation of the mental phenomena of certain forms of insanity, but up to a point it is a proved scientific fact. At what part or parts of the apparatus the interruption occurs is subject for further enquiry, but, if we may conjecture from the rapid results of research during the last few years, we may look hopefully to a not very remote future for important developments bearing on physiology, and, consequently, on medical psychology. Further, physiological observation has succeeded in breaking down the old idea of the action of the mind on the body. There is a wide difference between the conception of the action of the *organ* of mind on the body and the popular idea that the mind acts directly on other organs. We no longer, for instance, regard failures of function of the abdominal viscera in the insane as due to mental action, but as due to the impairment of the cerebral trophesial function which occurs coincidently with the appearance of mental symptoms.

Again, further, our whole conception of the nature and causes of acute insanity has been modified, I might almost say revolutionised, by the gradual advance in our knowledge of the action of toxins upon the structure and functions of the nerve-cell, and especially of the cortical cell. There is now the strongest evidence of toxæmia in certain forms of acute insanity and of the rapid implication of structure as a result of these poisonous agents. We are being forced to the conclusion that the mental symptoms in the acute psychoses are manifestations of a toxic physical condition, just as the delirium of fever is a symptom of certain phases of intoxication of the nervous system.

In fact, I may say that there is no development of medicine which has not been brought to bear on the subject, and with important results.

Granted what has been said as to changed views on insanity, the question arises what bearing has that change

had on the treatment of the various conditions comprised under that generic term. I hold that it emphasises and gives reason for the belief that the insanities must be submitted to treatment in their earliest stages, reasons which can be adduced before the legislature. This, of course, is true of all diseases, but in regard to the insanities the position is complicated by the false conception of their nature, still to some extent existent, and accordingly the public and the legislature do not yet regard them as the symptoms of corporeal disease. Rough asylum statistics showing the relation of recoveries to the period of incidence of symptoms have not carried conviction to the general public, and accordingly the tenor of our laws is towards the *protection* of those mentally afflicted rather than towards measures calculated to procure the cure of sick persons.

It is on these general grounds that I have urged on the Government the appointment of a Royal Commission to enquire into the subject in all its relations, and to consider whether a change in the Lunacy Laws is or is not demanded in the public interest.

The law under which we now work in England is a strictly lawyer's act. It was constructed without reference to any medical authority whatever. Neither medical Commissioner in Lunacy, nor Chancery Visitor was consulted. The leading idea was the protection of the public and the lunatic. No provision was made for the early treatment of patients. There was certainly allowed the certificate of emergency, but this was grudgingly admitted, and in at least one, if not two, Lunacy Amendment Bills, it was proposed that the term should be shortened. About the time the main Bill was introduced there had been one or two "scares" which had been most unjustifiably "written up" by certain papers. As I have often said the Bill was conceived in suspicion and born in panic. It contained one good provision and one ridiculous one, but taken over it all it was bound together by such an amount of hampering red tape, that its action has been found cumbrous and irritating. The clause I have designated as ridiculous is the one by which a person certified to be insane by two medical practitioners and com-

mitted to an asylum, may be submitted to the opinion of a justice of the peace, which determines the position. To this clause we may apply the *ex pede* line of argument, for it gives expression to the feeling of suspicion which permeates the whole Act. It was asserted by the promoters of the Bill that it was founded on the Scottish system. It did so, so far as the method of certification was concerned, but it took no account of two of the most important features of the Scottish Acts, viz., adequate power of inspection, and liberty to medical practitioners to treat incipient and early cases without certification. I have argued elsewhere that the liberty granted to us in the North cannot be accorded to a country in which the inspecting power is ludicrously insufficient. I am not going to trouble you with statistics, but will merely state that in Scotland that power is more than ten times greater than in England. As a consequence there is in that country a feeling of security in the public mind, without which we could not exercise the power conferred on us. As to its working, all I can say is that Clause 13 of the Scottish Act of 1866, by which we can treat cases in private houses for six months under a certificate of a medical person, and without reporting to the Commissioners, has worked smoothly for forty years, and that in no single instance has the slightest suspicion of abuse arisen.

The first step towards lunacy reform in England is a considerable increase in the number of Commissioners. The English Commissioners, in their last report, urge this strongly on the Government, in order that they may be able to perform adequately the ordinary duties of the office. I urged this point as strongly as I could when speaking on the Lunacy estimates, and pressed hard for the appointment of a commission to deal with the whole matter. The Attorney-General in his reply gave me no hope, and appeared to be satisfied with existing conditions. Within a week, however, he introduced a small Bill which proposed to grant to England a modification of Clause 13 of the Scottish Act of 1866. But it avoided the main issue, and it would have been my duty to have opposed the Bill if it had reached the second reading stage—which it did not—on the ground that it was merely a sop to Cerberus.

I want, however, to take up a much broader issue. The Scottish clause I have been speaking of mainly applies to the monied classes. What are we to do for the poor and for those of moderate means? We all know that incipient cases derived from this class cannot be submitted to treatment till symptoms are so pronounced as to warrant certification, that is to say, till such time as morbid action has made such advance as to retard recovery or prevent it altogether. *To the accumulation of unrecovered insane persons* the numerical increase of lunatics (I use the technical term) is mainly due. It is not entirely due to this cause, for there are others, such as the Government grant, the consideration of which can hardly be brought within the scope of this address. I know I can appeal to the experience of many here present that recovery is not difficult to obtain if treatment is applied during the initial stage, and that such treatment is most satisfactory in a large proportion of cases, inasmuch as not only is recovery from the immediate attack affected, but the tendency to recurrence is weakened. In fact, the cure often becomes complete, in that morbid action on the tissues having been arrested, and full opportunity having been given for their regeneration, no permanent solution of continuity remains.

Can analogous methods of treatment be provided for the insane of the poorer classes? That it can, in the opinion of those best able to judge, is amply set forth in the report of the committee of the London County Council appointed in 1890, at the instance of Mr. Brudenell Carter, to consider the propriety of erecting a hospital for the insane. The principal finding of the committee, founded on "the opinion of the most eminent and most experienced members of the medical profession," was to the effect that it was desirable that "an adequately equipped hospital, containing 100 beds, for the study and curative treatment of insanity, should be established in the Metropolis." Why and wherefore, and under whose influence this modest scheme was not carried into effect, I do not care to consider at present. As a matter of fact the report was pigeon-holed, and has not affected in any way the policy of the Council, except to the extent that

powers to erect "reception wards" were asked for in a Bill which never reached the second reading.

Glasgow has the honour of being the first city in Great Britain to carry such a scheme into effect. In 1890 one parish, the Barony, instituted what were termed observation wards for the reception of so-called "nervous cases." The building was by no means well adapted for the purpose, and the general arrangements were not satisfactory from a hospital point of view. Nevertheless, the results of the experiment were generally satisfactory.

The second stage of the experiment was undertaken in 1899, when two of the largest parishes of Glasgow were amalgamated under the Parish Council, which is also the Lunacy Board, and has the supervision of all lunatics within its area. It was conducted under somewhat improved conditions in wards set aside for the purpose in one of the city hospitals. Between 1899 and June of 1904, 1,345 persons were admitted, of whom 183 were sent to asylums, 86 died, and 1,052 were discharged "recovered" or "relieved." Satisfied with these results, in June 1904, the Parish Council proceeded to the third stage of the scheme. It erected wards for the special purpose, a pavilion attached to one of its general hospitals. This institution is situated in one of the most populous parts of the city. It contains 50 beds equally divided between the sexes. These wards are simple in structure and furnishings, they are in fact ordinary hospital wards, with a few side-rooms. They have no special appliances, such as padded rooms. They have been in operation since June of last year, and between that date and December 1904, 260 persons were admitted, of whom 13 died, 62 were at once sent to asylums, and 155 were discharged "recovered" or "relieved." I cannot submit any statement as to cost of erection or of maintenance, as the accounts for the current year are not yet closed. Those are the bare figures. But I am never content with bare figures, and accordingly I recently paid two prolonged visits to these wards, under the guidance of Dr. Carsewell, the certifying officer of the Parish Council, under whose charge the institution is placed. On both occasions I found forty patients under treatment. With one

or two exceptions they were evidently drawn from the poorest class, such as one finds in pauper asylums. Of these one-fourth were suffering from alcoholism in one or other of its phases; one man was convalescent from violent alcoholic mania; the remainder were the subjects of more or less intense delirium, whose treatment in an asylum or general hospital would have been necessary had the reception wards not been in existence. I emphasise this fact because it has been loosely stated that these wards are chiefly devoted to "drunks." That is not so, for of the total number admitted since 1890, only 28 per cent. were alcoholics. The symptoms of the other cases in hospital at the dates of my visits were referred to the usual classes of insanity as set forth in asylum reports. The average length of residence is twenty-five days, but it may be extended to six weeks. In a word, the class of cases was, allowing for social position, of the same type as we meet with in private practice, and which we treat either to a successful termination at their own homes, or in private dwellings, or relegate to asylums if unfitted for such treatment. This hospital provides for the poor a method of early curative treatment, which elsewhere can only be vouchsafed to the rich. I have gone thus far into details in order to show that the feasibility of a system by which the curative treatment of the insane poor during the earlier stage of the condition has been reduced to demonstration. It is the only institution of the kind in Great Britain. The only subject for regret is that it is not so fully utilised as it should be as a clinique for the students of the Medical School of Glasgow, for in its wards the insanities can be demonstrated as they occur in practice, at the period when the practitioner has to deal with them. Clinical instruction in an asylum is all very well in its way, but it is not worth argument to show the infinitely greater advantage that would accrue to all students were such wards open to him.

Let us look to what is done abroad. To each one of the twenty Universities of Germany a psychiatric clinic is attached, under the superintendence of the Professor of Psychiatrie. The buildings are either independent, in the neighbourhood of a general hospital, or they are separate

blocks of a hospital, or they are wards specially devoted to the purpose. In many instances they have been in existence for long; the clinic at Heidelberg, for instance, was established in 1878. They vary in size; Wurzburg has 60 beds; Tübingen, perhaps the most perfect institution of the kind, holds 120 beds. All alleged to be mentally unsound pass through these hospitals; if the nature of the case demands certification it is passed on to the asylum; the rest, consisting of early or mild cases of insanity, neurasthenia, the subjects of delirium due to fever or consequent on such diseases as pneumonia, post-operation cases, or neurasthenics—in fact, all members of the class which demands observation and treatment are retained without certification and treated to a termination without being reported to the State Office. These clinics are exactly on the same footing as the other clinics, medical and surgical, existing in all German Universities. If not to such a full extent as in Germany, similar provision is made in Austria, Italy, Switzerland, and the United States. Serieux puts it well when he says: "The beneficial influence exercised by the twenty University Clinics of Germany is easy to realise. They have given a vigorous impulse to the study of mental diseases; they form the scientific centres or nurseries for the proper education of the medical profession in psychiatry, and they present an ideal at which the provincial asylums should aim, viz., to become hospitals for the treatment of diseases of the brain."

I venture to think that my main contention is proved by the facts adduced. In most European countries and in Scotland the treatment of recent and acute insanity can be conducted in accordance with law under conditions best suited to procure rapid recovery. In England such is not the case. As regards the treatment of patients derived from the richer sections of the community, the practitioner has to sail very near to the wind of the law. I confess that on several occasions when treating cases south of the border I have deliberately broken the law, and, should occasion arise, I shall repeat the offence in the interest of my patient. We in England and Wales are hampered by a

law which prevents us in many instances from doing our duty to the opulent victim of insanity.

From a numerical point of view this is of comparatively small importance. The larger question applies to the accumulation of pauper lunatics in asylums, consequent on their not being submitted to treatment during the period in which morbid conditions are readily amenable to curative influences. If we are to learn anything from the experiment conducted in Glasgow, it is to the effect that it is highly desirable that all cases reported to the authorities as presenting mental symptoms should pass through the sieve of such an institution as there exists. The medical officer in charge can discriminate during a longer or shorter period of observation between those requiring transmission to an asylum, those who can be relegated to a general hospital, those who can be boarded out in private dwellings, and those who can be treated in the special hospital.

The public must be brought to see that the medical is closely connected with the fiscal position. At present it believes that it has done its duty in providing treatment in asylums, although it sorely resents the constantly increasing expenditure. If we can force in on the public mind that early treatment means economy we will soon gain sympathy and support. In all probability an amending Bill will be introduced this session, and, if the members of this Society, independently and collectively, make their views known to the Legislature, the first step towards reform will have been taken. There exists a substantial number of members of Parliament who would support the demand for a Commission to enquire into the whole subject, and not a few who would generally interest themselves in the general question. But the application must be supported by the leading members of the medical profession, by the leading scientific societies, and, if possible, by the Royal Colleges of Physicians and Surgeons. In the event of a Commission not being acceded to by the Government, on the introduction of an amending Bill, pressure should be applied in order to obtain provisions:—

(*a*) For the increase of the medical Commissioners.

(*b*) For the introduction of clause 13 of the Scottish Act of 1866.

(*c*) For the granting of power to County Councils to erect institutions for the early treatment of patients of the poorer classes, either in connection with or independent of general hospitals.

(*d*) For the granting of power to County Councils or Parish Councils to make provision for the after-treatment of patients discharged from asylums recovered or improved. I have not alluded to the last suggested provision in the body of this address, but I feel sure it will commend itself to the Society, as it is manifest that after-care would reduce the liability of such persons to recurrence of the attack.

Gentlemen, I have spoken in very general terms. You could not but notice that I could have elaborated my arguments in various directions. This I should have done before a popular audience, but I deemed it unnecessary to do so when addressing this Society. Indeed, I have restricted the length of my address in order to give time for discussion and criticism, in the hope that suggestions may be offered as to what action, if any, the Society can take to promote reform. I venture to suggest the appointment of a committee to watch legislation, and with powers to enlist the collaboration of bodies or persons interested in the question. In so doing I believe the Society would be conferring a great benefit on society at large, not merely by advocating procedure which would reduce expenditure, but, by what is of much higher importance, by pressing on the public measures which could not fail to stay the constantly increasing accumulation of chronic lunatics.

ON THE INTRINSIC FIBRES OF THE CEREBELLUM, ITS NUCLEI AND ITS EFFERENT TRACTS.

BY R. H. CLARKE, M.B.

AND

SIR VICTOR HORSLEY, F.R.S., F.R.C.S.

From the Laboratory of Chemical Pathology, University College London.

INDEX.

(1) General Introduction.
(2) Historical Introduction.
(3) Anatomical Introduction.
(4) Results.
(5) Summary.
(*a*) Relation of the cortex cerebelli to the cerebellar peduncles.
(*b*) Relation of the cortex cerebelli to the nuclei cerebelli.
(*c*) The arcuate system.
(*d*) Size of the intrinsic fibres of the cerebellum.

(1) GENERAL INTRODUCTION.

WE desire at the outset to express our acknowledgments to Dr. T. Woulfe Flanagan for much kind help in the preparation and execution of this work.

The cerebellum, both in its structure and function, has been the subject of much valuable research during recent years at home and abroad.

The anatomy of its lobes and main tracts, their relationship to each other, and the effect upon function of the removal or injury of each of them, have been established on a sound basis.

Further progress must largely depend upon a fuller knowledge of its minute structure; and the series of experiments set forth in this paper were undertaken to obtain more precise data on the true anatomical relations of the cortex of the cerebellum with its own and neighbouring nuclei, and on the actual origin of the efferent tracts.

Two other questions were kept specially in view :—

(1) Is there a direct efferent tract from the cerebellum to the spinal cord ?

(2) And if such a tract really exists, is it derived from the cortex or from the nuclei ; and, in the latter case, from which nuclei ? (*cf.* Thomas).

Until the introduction of Marchi's method the solution of all these questions was impossible, as neither the Weigert-Pal, nor any other method, is sufficiently delicate to distinguish the degeneration of individual fibres, or of small tracts. However, since Marchi's method came into general use relatively few experiments have been directed to the differentiation of the cerebellar cortex from its neighbouring nuclei, and of these none have been carried out with sufficiently minute precautions to make the results absolutely definite. Thus in Thomas's admirable work *Le Cervelet*, records of forty-two degeneration experiments on the cerebellum are given. The animals died in nearly half the cases, and in eighteen they lived less than ten days, while in a large number adventitious lesions were noted. Similarly, in Probst's valuable paper, in no case apparently was the lesion strictly confined to the cortex cerebelli. It is very difficult and sometimes impossible to avoid all accidental injuries, especially such as are due to slight bruising or strain of the peduncles, but the results obtained in such cases ought to be accepted with reserve, and only admitted in corroboration of those where no such mischances have occurred. We would also suggest that when an animal succumbs within a shorter period than three weeks of the operation, the absence of visible degeneration should not be accepted as definite proof that none exists ; since for adequate demonstration of degeneration three weeks is approximately requisite in the majority of animals.

In animals which have not survived more than a fortnight after an operation it is common to find that regions, where extensive degeneration must certainly exist, show very little or none at all. The negative evidence depending on the complete absence of stained fibres in a particular region is conclusive in a well-stained specimen, but is worthless in the

case of an animal which has not survived a lesion more than a fortnight. And, again, not only suppuration but any grave constitutional symptoms which may be connected with inflammatory changes near the lesion should, we think, render conclusions drawn from the presence of degenerate fibres of little value, unless these conclusions are very clearly corroborated by other evidence.

We have endeavoured to avoid such sources of error as far as we were able. The animals whose cases we have accepted as available for this paper were all kept three weeks before they were killed under chloroform; they were well nourished and in good health; the operation wounds all healed at once, and required no second dressing. Chloroform was employed for the operations, which were performed by one of us (V. H.), and the animals were always kept fully anæsthetised. The central nervous system was examined after fixing in Müller's fluid and staining in Busch's modification of Marchi's fluid, by being cut in serial sections. By our experiments we have accumulated sufficient data to justify a positive answer to the questions proposed.

From twenty-three uncomplicated lesions (in seventeen cats, two dogs, four monkeys, Macacus Rhesus) of the lateral lobes, and of the anterior and posterior vermis the nuclei being absolutely uninjured in thirteen cases, we have obtained well-marked and well-stained degenerations.

All the specimens show abundant arcuate fibres connecting various parts of the cortex, and other fibres passing to the nearest cerebellar nuclei, whether roof, globose, or dentate, but none going directly from the cortex cerebelli to any of the peduncles. Our series of sections also show that all efferent cerebellar cortical fibres in the first instance pass to one or other of the central nuclei, and principally to the one which is nearest to their origin.

(2) Historical Introduction.

The literature of research on the cerebellum, both anatomical and physiological, is already voluminous.

Yet hitherto but few definite facts have been obtained on the special object of our investigation. The reasons for this have been already fully explained.

The following is a list, chronologically arranged, of the chief authorities on the cerebellum who have preceded us, with a brief summary of their results on the special points referred to.

(I.) *Marchi* (1891-1892), to whom belongs the merit of devising the valuable method by which many observers, including ourselves, have obtained their results, was led at the beginning of the investigation of this complex subject to take the view that lesions of the cerebellum (*i.e.*, cortex and nuclei together) caused degeneration of descending tracts from the cerebellum to the spinal cord. Subsequent research has proved that such descending degenerations were the consequence, not of lesions of the cerebellum, but of parts of the mesencephalon and bulb.

(II.) *Ferrier* and *Turner* (1894).—These authors employed the Weigert-Pal method of staining, and were therefore precluded from observing the precise connection between the cortex and the nuclei of the cerebellum. They arrived, however, at the conclusion that injury of Deiter's nucleus was necessary to evoke any degeneration of descending tracts in the spinal cord.

(III.) *Risien Russell* (1894-1896).—The object of Dr. Russell's researches was the determination of the relations between the cerebellum as a whole and the rest of the cerebro-spinal axis. To this end the lesions were designedly extensive, and, consequently, involved the nuclei of the cerebellum. He employed the Marchi method, and established on an irrefragable basis the conclusion that no direct paths exist between the cerebellum and the spinal cord.

(IV.) *Biedl* (1895).—The degenerations observed by Biedl in the spinal cord in his experiments were certainly due to the influence of the lesion on neighbouring portions of the bulb, and, therefore, although his work appeared to support the original idea of direct connection between the cerebellum and the spinal cord, he does not regard it now as in fact doing so.

No.	Lesion.	Diagram. Shaded area indicates lesion, for further explanation see p. 19.
Rhesus 22 ...	(1) Superficial removal of uvula and bruising of pyramid, and slight bruising of posterior folium of nodulus. (2) Deep destruction of uvula fibres and of nucleus fastigii; bruising of nucleus globosus on right side. [Nucleus globosus and fastigii on left side normal.] Bruise of nucleus cuneatus, right side.	See photograph, Plate VII. ...

No.	Lesion.	Diagram. Shaded area indicates lesion, for further explanation see p. 19.
Rhesus 23 ...	(1) Eight folia of lobus quadrangularis, mesial third. (2) Neighbouring border of corresponding eight most posterior folia of the culmen.	See photograph, Plate VII. ...

r further	Nuclei.			
	Dentatus.	Globosus.	Fastigii (vel Tecti).	
... ...	Nil	Fine fibres to right nucleus globosus.	Very numerous fine fibres to upper part of nucleus fastigii, which is extensively destroyed on right side.	C

Additional Case of Lesion, lim

r further	Nuclei.			
	Dentatus.	Globosus.	Fastigii (vel Tecti.	
... ...	Some fine fibres to the inner aspect of the posterior pole.	Numerous fine fibres to inner border.	Numerous fine fibres to outer border	N

ther	Nuclei.				Peduncles.			Inferior Olive.	Spinal Cord.
	Dentatus.	Globosus.	Fastigii (vel Tecti).	Vestibuli.	Superior.	Middle.	Inferior.		
...	Nil	Fine fibres to right nucleus globosus.	Very numerous fine fibres to upper part of nucleus fastigii, which is extensively destroyed on right side.	Coarse fibres to the nuclei vestibuli of both sides.	Both superior peduncles contain fibres going to red nucleus.	Nil	A few fibres in restiform; *cf.* bruise of nucleus cuneate.	Nil	Nil ...

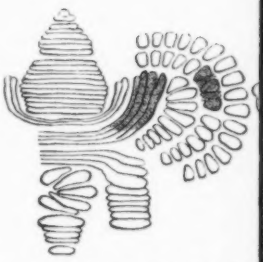
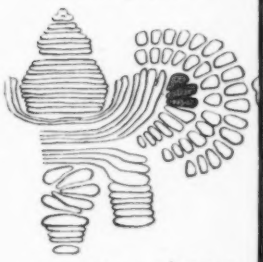
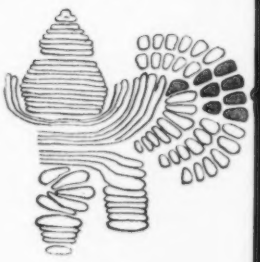
Additional Case of Lesion, limited to Cortex of Lateral Lobe.

ther	Nuclei.				Peduncles.			Inferior Olive.	Spinal Cord.
	Dentatus.	Globosus.	Fastigii (vel Tecti.	Vestibuli.	Superior.	Middle.	Inferior.		
...	Some fine fibres to the inner aspect of the posterior pole.	Numerous fine fibres to inner border.	Numerous fine fibres to outer border	Nil	Nil	Nil	Nil	Nil	Nil ...

	Peduncles.			Inferior Olive.	Spinal Cord.	Arcuate Fibres.	Remarks.
Vestibuli.	Superior.	Middle.	Inferior.				
oarse fibres to the nuclei vestibuli of both sides.	Both superior peduncles contain fibres going to red nucleus.	Nil	A few fibres in restiform; *cf.* bruise of nucleus cuneate.	Nil	Nil ...	The arcuates could not be determined on account of the other lesions.	Fine fibres to right paraflocculus and flocculus, coarser fibres to left paraflocculus and flocculus from right nucleus fastigii. Large number of coarse fastigio-coerulean fibres on right side only. The large majority of coarse fibres from nucleus fastigii, which are very numerous in anterior cerebellar commissure, end in side of bulb just external to 6th nerve.

ited to Cortex of Lateral Lobe.

	Peduncles.			Inferior Olive.	Spinal Cord.	Arcuate Fibres.	Remarks.
Vestibuli.	Superior.	Middle.	Inferior.				
il	Nil	Nil	Nil	Nil	Nil ...	Fine arcuates only extend in each direction as far as the adjacent folium, and nowhere reach anterior part of culmen or lobus centralis.	

No.	Lesion.	Diagram. Shaded area indicates lesion, for fur explanation see p. 19.
	Lateral Lobe or "Hemisphere." (1) *Anterior Pennate Lobule.* (2) *Posterior Pennate Lobule.* (3) *Flocculus and Paraflocculus.* (4) *Supero-anterior region of the whole cortex cerebelli.*	
Cat 1. ...	Four folia of left anterior pennate lobule. The margin of the lesion involves the edge of the dorsal paraflocculus at the pole of the lateral lobe.	 See photograph, Plate I.
Cat 2 ...	The three outermost folia of the posterior pennate lobule.	 See photograph. Plate I.
Cat 3 ...	Slight bruise of *flocculus*, *paraflocculus*, and the tip of the posterior pennate lobule (one folium).	

for fur'er
19.

ate I.

te I.

Table I.—Results of the Experiments in the Present Research. (Degenerations Observed.)

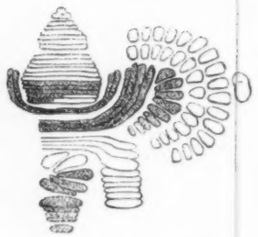
Nuclei.				Peduncles.			Inferior Olive.	Spinal Cord.
Dentatus.	Globosus.	Fastigii (vel Tecti).	Vestibuli.	Superior.	Middle.	Inferior.		
Numerous fine fibres to and in the outer border.	Nil	A few fine fibres in homolateral nucleus	Nil	Nil	Nil	Nil	Nil	Nil
A few fine fibres in the outer side.	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Nil	Nil	Fine and coarse fibres to ventral surface	Nil	Nil	Nil	Nil	Nil	Nil

Table I.—Results of the Experiments in the Present Research. (Degenerations Observed.)

for further 19.	Nuclei.				Peduncles.			Inferior Olive.	Spinal Cord.
	Dentatus.	Globosus.	Fastigii (vel Tecti).	Vestibuli.	Superior.	Middle.	Inferior.		
	Numerous fine fibres to and in the outer border.	Nil	A few fine fibres in homolateral nucleus	Nil	Nil	Nil	Nil	Nil	Nil
te I.	A few fine fibres in the outer side.	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
te I.	Nil	Nil	Fine and coarse fibres to ventral surface	Nil	Nil	Nil	Nil	Nil	Nil

Results of the Experiments in the Present Research. (Degenerations Observed.)

Nuclei.		Peduncles.			Inferior Olive.	Spinal Cord.	Arcuate Fibres.	Remarks.
Fastigii (vel Tecti).	Vestibuli.	Superior.	Middle.	Inferior.				
A few fine fibres in homolateral nucleus ...	Nil	Nil	Nil	Nil	Nil	Nil ...	*Arcuates.*—A few fibres ascend to *pyramid* and lowest two folia of *para-median lobe*. A few fibres ascend to *culmen* and *lobus centralis*. A few cross to opposite nucleus, fastigii and round bases of folia, posterior to primary fissure. Fine fibres pass down to the *paraflocculus*. A few of the fibres thus passing to the principal parts of the vermis cross the mid line.	
Nil	Nil	Nil	Nil	Nil	Nil	Nil ..	Nil	Fine fibres pass across the medulla of the *vermis*, but none in front of the commissure. A few fibres pass to the lateral region of *corona*, *i.e.*, to the folia of the pennate gyri.
Fine and coarse fibres to ventral surface ...	Nil	Nil	Nil	Nil	Nil	Nil ...	*Arcuates.*—Very few fine fibres to folium immediately adjacent to slightly bruised folia.	The fibres described as entering the *nucleus fastigii* run straight inwards along the roof of the *recess* of *Retzius*.

No.	Lesion.	Diagram. Shaded area indicates lesion, for further explanation see p. 19.	Nuclei.				Peduncles.			Inferior Olive.	Spinal Cord
			Dentatus.	Globosus.	(Fastigii vel Tecti).	Vestibuli.	Superior.	Middle.	Inferior.		
Cat 4 ...	Bruising and laceration of surface of all folia of *lateral lobe*, excepting the posterior pennate and posterior paraflocculus lobules.		Many fine fibres... ...	Some fine fibres... ...	Very few fine fibres	Many fine fibres... ...	*Left.*—A few coarse fibres.	Nil	Nil	Nil	Nil
	Combined Lateral Lobe and Vermis Lesion. (5)										
Cat 5 ...	Two parts:— I.—*An upper horizontal incision* which separates (*a*) *on left side* the cortex of five folia of the *culmen*, and also of the lateral lobe. On (*b*) the *right side* the lesion extends horizontally to the same distance, *i.e.*, to the dorsal paraflocculus, but sparing in part the two most caudal folia of culmen. II.—*An oblique incision* through the pyramid into the fourth venticle, completely detaching the uvula, nodulus, and ventral half of the pyramid.	 *Note*—Lesion of left lateral corresponds to above. See photograph, Plate II.	Nil	Nil	I.—*Right.* Posterior third full of fine degeneration. II.—*Left.*—Lower third ploughed up by lesion ii. A large number of coarse fibres pass between nucleus and the anterior commissure and the right nucleus fastigii. There are also fibres which pass down on inner side of superior cerebellar peduncle, reaching the gray matter around the peduncle. Some fibres are directed to the substantia reticularis of the *bulb*, just ventral to the motor nucleus of the Vth nerve. In travelling forwards they pass among the fibres of the outer region of the pars reticularis, and especially between the pallio-tectal fibres, where these are about to enter the right lateral fillet nucleus. They end in the gray matter round the right superior cerebellar peduncle and in the central gray matter of the aqueduct.	Fibres to Deiter's nucleus.	Nil	Few fine fibres to right pontine nucleus.	Nil	Few fine fibres to right olive.	Nil
	Vermis (anterior) Lesions, specially including the Culmen. (6) *Culmen.* (7) *Culmen and all the middle lobe.* (8) *Culmen: lobus centralis and first two lobules of middle lobe.*										
Rhesus 6 ...	The lesion consists of a horizontal puncture entering the culmen close to the mesial plane just in front of the primary fissure, undermining the five posterior folia of the culmen. The puncture in its greatest depth reaches just to the dorsal limit of the central white mass of the lobe.	See photograph, Plate II.	Nil	Numerous fine fibres on dorsal surface.	Very numerous fine fibres	Nil	Nil	Nil	Nil	Nil	Nil

estibuli.	Peduncles. Superior.	Middle.	Inferior.	Inferior Olive.	Spinal Cord.	Arcuate Fibres.	Remarks.
fibres... ...	*Left.*—A few coarse fibres.	Nil	Nil	Nil	Nil ...	*Arcuates.*—Heavy degenerated fine fibres in dorsal and ventral divisions of *homolateral lobe.* I.—*Anteriorly* these fibres pass forwards between the nuclei, especially to central folium of *anterior vermis*, being limited to the same half (except a few which run from the opposite side), arranged in four bundles, the largest of which is close to the middle line. II.—*Posteriorly*, the fibres extend into the whole of the *posterior vermis*, except the uppermost three or four folia.	Fine fibres run to all the nuclei; those descending to nuclei vestibuli terminate there. Posteriorly, a few cross the *nuclei fastigii* in order to gain opposite side of *posterior vermis.* The collaterals in nucleus *dentatus* run antero-posteriorly.
Deiter's nucleus.	Nil ...	Few fine fibres to right pontine nucleus.	Nil	Few fine fibres to right olive.	Nil ..	*Arcuates.*—Fine fibres can be traced into the posterior part of the (left) paraflocculus; the lateral lobe and especially the posterior pennate lobule. There are more corticopetal fibres in the uvula than in the nodulus. Some pass into the uvula from the lesion in the nodulus two folia distant. There are fine fibres in the distal third of each of the folia of the anterior vermis, diminishing in number from above down.	In the folia, both ascending and descending fibres seen. These terminate in (1) *gray matter* round superior cerebellar peduncle; (2) tegmentum, dorsal of red nucleus; (3) *corpus recticulare* of bulb in the region of the nucleus magnocellularis; (4) Deiter's nucleus.
... ...	Nil	Nil	Nil	Nil	Nil ...	*Arcuates.*—In the degeneration of one of the injured folia, cut obliquely, coarse fibres of a hook shape (Ramon y Cajal) are seen, the convexity of the hook being near the granule layer. Fine degenerated fibres run forward in the white matter of the two adjacent ventral folia, as far as the cortex of the front of the vermis. Laterally fine arcuate fibres pass to the immediately adjacent folium only. A few fine fibres pass down into the main folium of the uvula. No fibres can be traced into the nodulus.	*Nucleus fastigii.*—A few fibres decussate in the *raphè* between the *nuclei fastigii*, and in the posterior bundles of the *anterior commissure.*

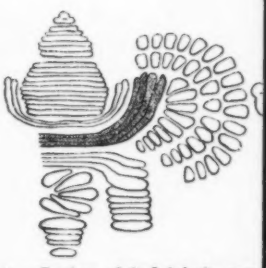
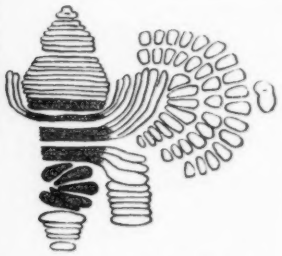
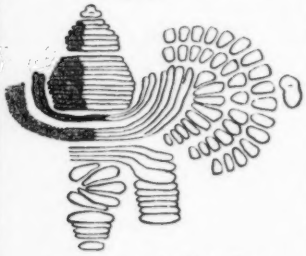
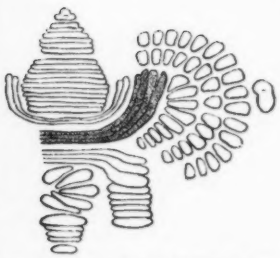
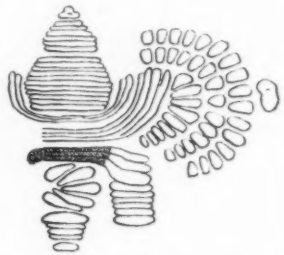
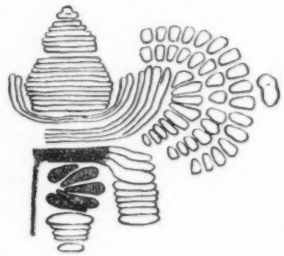
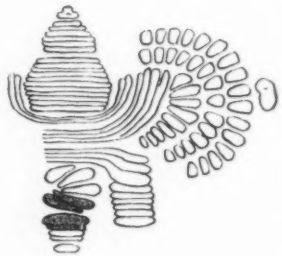
No.	Lesion.	Diagram. Shaded area indicates lesion, for fur explanation see p. 19.
Cat 7 ...	The lesion destroys the two posterior folia of the culmen, and all folia behind the primary fissure as far as the uvula. The lesion extends to *paramedian fissure* on each side.	See photograph, Plate III.
Cat 8 ...	The lesion involves the left paramedian region of the anterior vermis, including the two mesial folia of the lateral lobe. The lingula is uninjured. The outer third of the left half of each of the folia of the lobus centralis and culmen is destroyed, together with the left four-fifths of the first and second lobules of the middle lobe. **Vermis (posterior) Lesions.** (9) *Second Lobule of Middle Lobe and Anterior Pennate Lobule.* (10) *Third Lobule of Middle Lobe.* (11) *Third and Fourth Lobules of Middle Lobe.* (12) *Fourth Lobule of Middle Lobe (Pyramid).* (13) *Fourth Lobule and Uvula.* (14) *Fourth Lobule and Uvula.* (15) *Uvula.* (16) *Fourth Lobule Uvula and First Lobule of Middle Lobe.*	See photograph, Plate III.
Cat 9 ...	The lesion is an incision passing through and under cortex of the second lobules of the middle lobe to the right *lateral lobe*, as far as the fourth folium of the anterior pennate lobule, where it reaches the surface. The *left* side is equally injured.	 *Note.*—Lesion of 2nd lobule same on right side. See photograph, Plate IV.

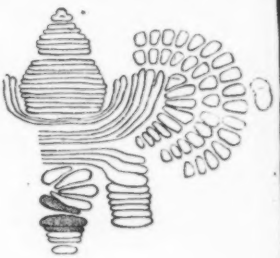
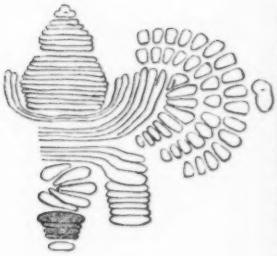
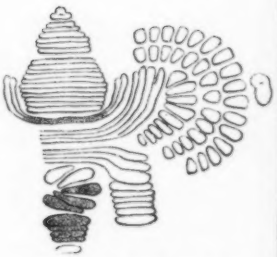
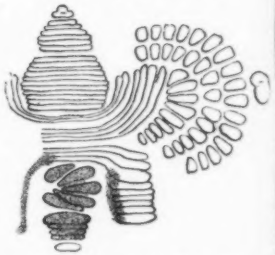
Diagram. Shaded area indicates lesion, for further explanation see p. 19.	Nuclei.			
	Dentatus.	Globosus.	Fastigii (vel Tecti).	Vestibuli.
 See photograph, Plate III.	Relatively few fine fibres	Very few fine fibres in dorsum, more *right* than *left*.	Crowded with fine degenerated fibres posteriorly.	A few fine fibres entering upper and posterior angle on left side.
 See photograph, Plate III.	Fine fibres entering anterior and inner aspect of nucleus and crossing anterior commissure.	Fine fibres along upper anterior and mesial borders.	Fine fibres in upper, inner, and mesial parts. Nil in outer.	A few fine fibres in lower part of the left nuclei.
 *Note.*—Lesion of 2nd lobule same as on right side. See photograph, Plate IV.	Fine fibres	Fine fibres	Fine fibres	A few fine fibres, on both sides.

	Peduncles.			Inferior Olive.	Spinal Cord.	Arcuate Fibres.	Remarks.
Vestibuli.	Superior.	Middle.	Inferior.				
few fine fibres entering upper and posterior angle on left side.	Nil	Nil	Nil	Few fibres of doubtful origin in the right olive.	Nil ...	*Arcuates.*—Fine collaterals from the *nucleus fastigii* into the *lingula*. Fine fibres pass to the folia of the *lobus centralis* in front of the lesion, and from the lesion to the proximal folium of the *anterior pennate lobule*. Below the lesion there are a few fine fibres to the folia of the caudal half of the uvula.	Numerous fastigio-brachial fibres, *i.e.*, around superior cerebellar peduncle.
few fine fibres in lower part of the left nuclei.	Nil	Nil	Nil	Nil	Nil ...	*Arcuates.*—A very few fine fibres can be traced in the third folium from the outer border of the lesion; fibres from folia posterior to the primary fissure extend to, but not beyond, the paramedian sulcus: while in folia anterior to the primary fissure they do not extend beyond the middle line, except very scantily.	The lesion divides several folia transversely. These show both ascending and descending degenerate fibres, of which the descending are coarser than the ascending.
few fine fibres, on both sides.	Nil	Nil	Slight bruise of *left*.	Nil	Nil ...	*Arcuates.*—A great many fine fibres in the white fibres of the *anterior vermis*, but no fibres pass to the lingula. Extremely few fine fibres pass to the right paramedian lobe, and scarcely any to the posterior pennate lobule.	Some coarse fibres in the *anterior commissure* were seen to be coming from the inferior peduncle, and traceable to the nucleus fastigii and nucleus dentatus.

No.	Lesion.	Diagram. Shaded area indicates lesion, for further explanation see p. 19.	Nuclei.		
			Dentatus.	Globosus.	Fastigii (vel Tecti).
Cat 10 ...	Three folia of the third lobule of the middle lobe of the vermis. The lesion involves the whole breadth of the vermis.	 See photograph, Plate IV.	Nil	Nil	Very scanty fibres run to mesial surface ...
Cat 11 ...	The lesion destroys the third and fourth (pyramid) lobules of the middle lobe of the vermis, extending across its whole breadth. There is slight bruising of the mesial border of the left *paramedian lobe.*		Few fibres in the *left* and very few in the *right.*	Very many fibres in the *left.* Few on the *right.*	Very many fibres. *Left* more than *right* ...
Rhesus 12 ...	Strictly confined to the dorsal surface of three folia of the pyramid.	See photograph, Plate V.	Nil	Nil	Numerous fine fibres ... ...
Cat 13 ...	Bruising of two adjacent folia of the uvula and pyramid.		Nil	Nil	Fine fibres proceeding to dorsum of nucleus...

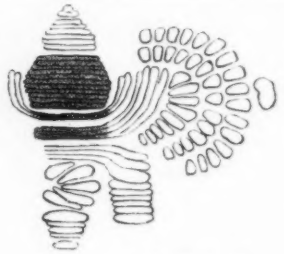
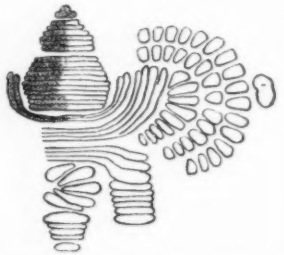
her	Nuclei.				Peduncles.			Inferior Olive.	Spinal Cord
	Dentatus.	Globosus.	Fastigii (vel Tecti).	Vestibuli.	Superior.	Middle.	Inferior.		
	Nil	Nil	Very scanty fibres run to mesial surface ...	Nil	Nil	Nil	Five or six fibres observed due to local bruising.	Nil	Nil ...
	Few fibres in the *left* and very few in the *right*.	Very many fibres in the *left*. Few on the *right*.	Very many fibres. *Left* more than *right* ...	Nil	Nil	Nil	Nil	Probably none. Determination obscured by concurrent lesion of fillet.	Nil ...
	Nil	Nil	Numerous fine fibres	Nil	Nil	Nil	Nil	Nil	Nil
	Nil	Nil	Fine fibres proceeding to dorsum of nucleus...	Nil	Nil	Nil	Nil	Nil	Nil ...

	Peduncles.			Inferior Olive.	Spinal Cord.	Arcuate Fibres.	Remarks.
Vestibuli.	Superior.	Middle.	Inferior.				
Nil	Nil	Nil	Five or six fibres observed due to local bruising.	Nil	Nil ...	*Arcuates.*—A few fine fibres pass to the folia immediately adjacent to the lesion on either side.	Nil.
Nil	Nil	Nil	Nil	Probably none. Determination obscured by concurrent lesion of fillet.	Nil ...	*Arcuates.*—The distribution of the arcuate fibres in this specimen is indeteriminate, because on the left side the inferior cerebellar peduncle was affected by another lesion.	A few *fastigio-brachial* fibres on *left* side.
Nil	Nil	Nil	Nil	Nil	Nil ...	*Arcuates.*—Extremely few fine fibres in the folium immediately adjacent to the lesion, above and below.	Nil.
Nil	Nil	Nil	Nil	Nil	Nil ...	*Arcuates.*—Fine fibres to the two anterior folia of the uvula. No fibres observed entering nodulus.	The fibres on the dorsum of nucleus fastigii pass close round the bases of folia of posterior vermis.

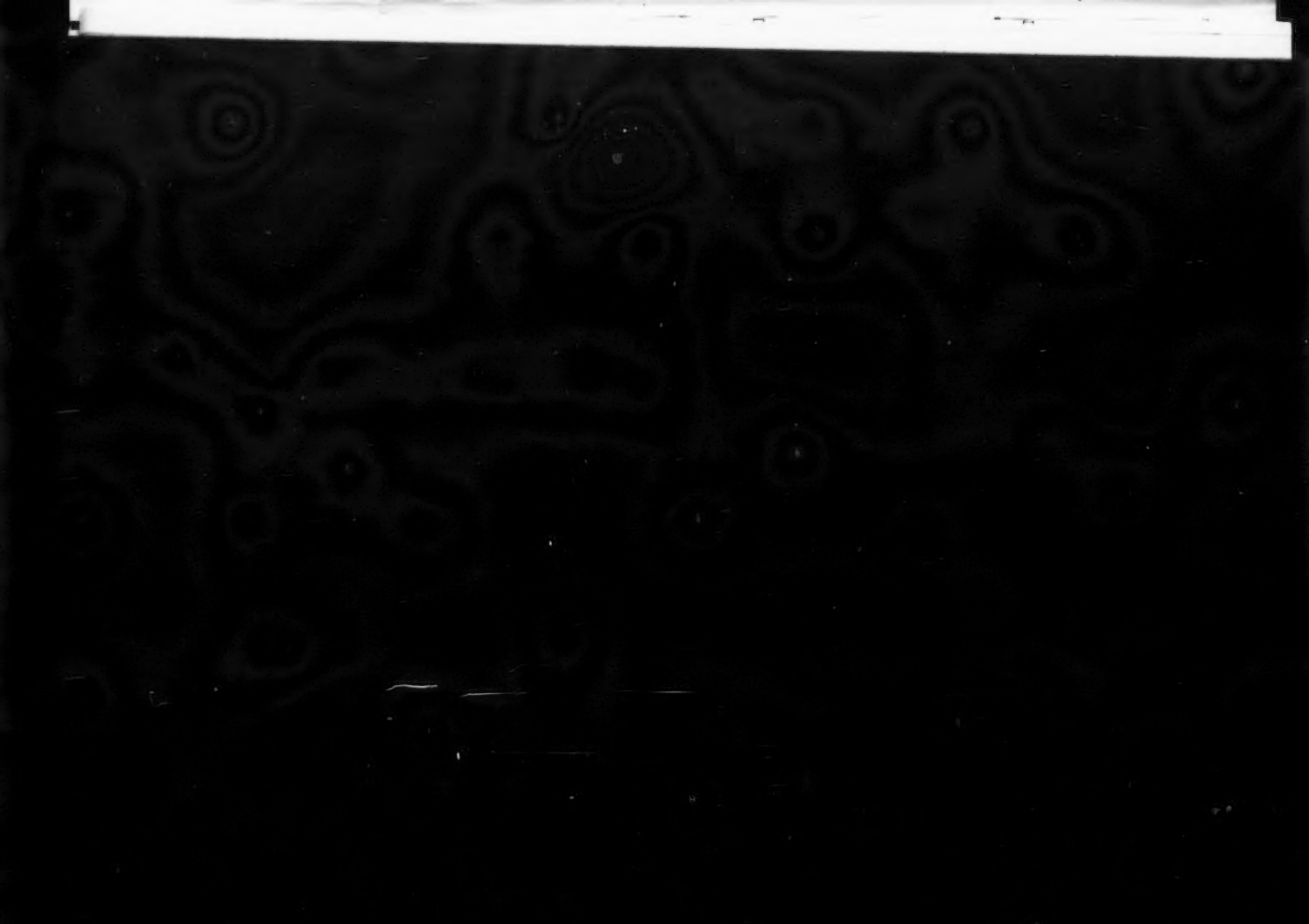
No.	Lesion.	Diagram. Shaded area indicates lesion, for further explanation see p. 19.
Cat 14 ...	Destruction of lowest folium of *pyramid*, and of two folia of uvula.	
Cat 15 ...	Destruction of the whole *uvula*	
Cat 16 ...	Nodule normal; folia of uvula and ventral half of pyramid destroyed; also white matter of two most anterior folia of the first lobule of the middle lobe.	
	Lesions of Cortex Cerebelli, complicated by injury of the Cerebellar Nuclei. (17) (18) (19) (20) (21)	See photograph, Plate V.
Cat 17 ...	The lesion causes destruction of right half of the pyramid, and undermines the remainder and the *uvula*. The lesion extends to the surface of the left *nucleus globosus* and to left *nucleus fastigii*. There is also slight bruising of the adjoining margins of the folia of each *paramedian lobe*.	
		See photograph, Plate VI.

further	Nuclei.				Peduncles.			Inferior Olive.	S
	Dentatus.	Globosus.	Fastigii (vel Tecti).	Vestibuli.	Superior.	Middle.	Inferior.		
	Nil	Nil	Many fine fibres to dorsum of nucleus	Nil	Nil	Nil	Nil	Nil	N
	Nil	Fine fibres	Medium and fine fibres	Nil	Nil	Nil	Nil	Nil	N
	Outer part nil	As in nucleus fastigii ...	Fine fibres in upper part of both nuclei. Numerous in right.	Nil	Nil	Nil	Nil	Nil	N
V.									
	Very few fibres reach the outside of nucleus dentatus on both sides.	Some fibres pass to the nucleus globosus on the right side.	*Right* contains degenerate fibres about the junction of its upper and middle thirds. *Left* has a few degenerated fibres in its middle zone.	Nil	Nil	Very few fine fibres.	Nil	Fine fibres pass as internal arcuate bundles to the right olive.	N
VI.									

Vestibuli.	Peduncles. Superior.	Peduncles. Middle.	Peduncles. Inferior.	Inferior Olive.	Spinal Cord.	Arcuate Fibres.	Remarks.
... ...	Nil	Nil	Nil	Nil	Nil ...	*Arcuates.*—To remainder of *uvula*, diminishing anteriorly; very few fine fibres seen in one section of *nodulus*; in other sections of nodulus, none.	The fibres to the nucleus fastigii pass close round bases of folia of *posterior vermis*.
... ...	Nil	Nil	Nil	Nil	Nil ...	Nil	*Nuclei.*—Medium sized and fine fibres pass to the dorsal borders of both nuclei fastigii; the medium occupy the raphè and appear to decussate therein; the fine fibres appear to pass to the nucleus globosus, but are obscured by precipitate.
... ...	Nil	Nil	Nil	Nil	Nil ...	*Arcuates.*—Although the lesion involves the left side of the *uvula*, no arcuate fibres were traced into the *nodulus*. Fine fibres traced into the two folia of the culmen immediately in front of the *primary fissure*. Also into the paramedian lobe the mesial border of which is injured. No arcuate fibres traced further, *i.e.*, to paraflocculus.	Coarse fibres ascending from the eighth nerve, accidentally injured, divide into two groups—one caudal (posterior) passing to the *dorsal vestibular nucleus*, traversing the ventral part of the *striæ acusticæ*. The anterior set divide in the gray matter on the *superior cerebellar peduncle*. After the bifurcation a few fibres ascend to the ventral anterior angle of the *nucleus fastigii*, the others pass towards the *dorsal tegmental nucleus* of Gudden.
... .	Nil	Very few fine fibres.	Nil	Fine fibres pass as internal arcuate bundles to the right olive.	Nil ...	*Arcuates.*—Fine arcuate fibres pass on the *right* side, as far as the *paraflocular region*; on the *left* side only to first two folia of the anterior pennate lobule. Numerous coarse fibres, arising from *nucleus fastigii* and from junction thereof with *nucleus globosus*, decussate in front of nucleus fastigii pass to and around superior cerebellar peduncle, some ending in gray matter on both sides of peduncle, while others run forward and pass through and around cerebral fifth root, to terminate in (*a*) *central gray*; (*b*) *locus cœruleus*; (c) gray matter of *substantia reticularis* (upper and outer). Most of this system is homolateral. The plane of highest extension forwards is that of the entry of the fourth nerve into the mesencephalon.	Nil.

No.	Lesion.	Diagram. Shaded area indicates lesion, for further explanation see p. 19.	Nuclei.				Peduncl	
			Dentatus.	Globosus.	Fastigii (vel Tecti).	Vestibuli.	Superior.	Middl
Cat 18 ...	The lesion is twofold ; as follows :— (*a*) Horizontal section, separating the first and second lobules of the middle lobe and all the folia of the culmen. (*b*) Vertical, which reaches through vermis to the posterior collicular commissure. The lesion involves the dorsal borders of the nuclei, viz., *fastigii* on both sides, *globosus* markedly on left, slightly on the right.	 See photograph, Plate VI.	A few fine fibres to outer part.	Many fine and medium fibres.	Many fine and medium fibres	Fine and Medium fibres on both sides, especially in Deiter's nucleus.	A few medium fibres	Very few fibres.
Dog 19 ...	Horizontal incision. Anterior limit, front surface of vermis. Lateral limit from the sides of vermis, extending on right side to lateral pole of hemisphere, extending on *left* side to middle of anterior pennate lobule. Posteriorly, incision enters and separates off posterior surface (apparently the junction of the 3rd and 4th lobules of the middle lobe) of cerebellum, reaching middle of *paramedian lobe* on each side. The ventral limit of the lesions is the posterior "commissure," and dorsal border of all the nuclei. The lesion extends into the substance of right nucleus fastigii.	... .	*Left.* — Fine collaterals, especially into inner two-thirds.	*Left.*—Very many fibres, but less numerous than in right.	*Left.*—Very many fibres, but less numerous than in right.	Many fibres coarse, medium, and fine.	*Left*, almost all the degeneration fibres are contained in the dorsal hook. *Right*, numerous fibres of various sizes.	Few fine of ind origin.
Cat 20 ...	The lesion destroys whole left *lateral lobe* and *flocculus*, encroaches on left half of *anterior lobe of vermis*, destroys *lingula*, penetrating left half of *bulb* opposite *pyramid* in *pons*. It destroys all the white matter of the lateral lobe. A separate laceration totally destroys *left nucleus fastigii* only.	 *Note.*—The whole left lateral lobe was destroyed by sub-cortical lesion.	Fine fibres from lesion enter the nucleus.	Fine fibres from lesion...	*Right*, normal, receives fine fibres from lesion of lateral lobe. *Left.*—Destroyed by lesion.	Destroyed on *left* side. Number of degenerated fibres of uncertain origin, pass towards its site.	*Right.*–Normal. *Left.*—Totally degenerated	Lesion e to rap pons.
Dog 21 ...	The lesion may best be described in parts. (1) Of left upper *paramedian sulcus* cutting off upper half of left *lateral lobe* completely, *i.e.*, both pennate lobules. (2) Continuous with, but at right angles to foregoing, cutting off the folia of the first two lobules of middle lobe of vermis. (3) Lesion also passes through both superior cerebellar peduncles. (4) Puncture of posterior border (lower third) of substance of *posterior colliculus*. (5) Injury of left *restiform body* low down.	... .	Many fine fibres... ...	Many fine fibres... ...	Many fine fibres	Many fine fibres... ...	*Left.*—Totally degenerated.	A few n fibres.

	Peduncles.			Inferior Olive.	Spinal Cord.	Arcuate Fibres.	Remarks.
Vestibuli.	Superior.	Middle.	Inferior.				
Fine and Medium fibres on both sides, especially in Deiter's nucleus.	A few medium fibres	Very few fine fibres.	Nil	Medium size fibres to outer end of olive.	Nil ...	*Arcuates.*—Extend as far outwards as the second folium of the lateral lobe on the left side.	
Many fibres coarse, medium, and fine.	*Left*, almost all the degeneration fibres are contained in the dorsal hook. *Right*, numerous fibres of various sizes.	Few fine fibres of indefinite origin.	Nil	Fine fibres to both olives.	Nil ...	*Arcuates.*—The extent of the lesion prevents accurate determination of the arcuate system.	As the lesion (q. v.) destroys part of the *nucleus fastigii* on the right side, it and its neighbours contain more degenerated fibres than on the left side. Large masses of fine fibres descend to *Deiter's nucleus* on each side, and a large number of coarse and medium fibres descend from both nuclei fastigii. *The fastigio-brachial* fibres are numerous; *fastigio-floccular*, few; *fastigio-coerulean*, intermediate in number. From these bundles of fibres, collaterals are given off opposite the base of *floccular peduncle* and the dorsal border of the *tuberculum acusticum*. These collaterals run inwards as far as the anterior limit of the seventh nucleus, around which they ramify in the substantia reticularis. They disappear at level of lower border of *tuberculum acusticum*.
Destroyed on *left* side. Number of degenerated fibres of uncertain origin, pass towards its site.	*Right.*—Normal. *Left.*—Totally degenerated	Lesion extends to raphè of pons.	Nil	Nil	Nil ...	*Arcuates.*—Fine fibres extend all through *vermis*, but not in right lateral lobe. Cortico-petal degeneration everywhere accompanies the destruction of white matter of the lateral lobe.	This case gives good proof that the middle peduncle is chiefly ponto-cerebellar. Inasmuch as, with complete destruction of the cortex of the *lateral lobe*, the degenerated fibres in the homolateral *middle peduncle* are those which definitely emanate from a small lesion in the pontine raphè. Strongly marked *fastigio-floccular*, *fastigio-brachial*, and *fastigio-coerulean* tracts are also present in this case.
Many fine fibres	*Left.*—Totally degenerated.	A few medium fibres.	Involved in lesion, contains numerous degenerated fibres.	Indeterminate owing to bulbar lesions.	Nil ..	The lesion is too extensive to permit of any accurate statement respecting the source of arcuate fibres.	*Nuclei.*—On right side, fine fibres pass in diminishing numbers from roof nucleus to dentate nucleus. All the nuclei are considerably damaged. The left *nucleus fastigii* is flanked by large masses of fine degenerate fibres. The *nuclei vestibuli* show on both sides very many fine fibres entering the upper pole and distributed all over the nuclei.



(V.) *Münzer* and *Wiener* (1895).—These authors also found no fibres proceeding directly from the cerebellum to the spinal cord.

(VI.) *Thomas* (1897).—The most extensive and thorough investigations on the present subject have been undertaken by Thomas. The general results he obtained may be summarised as follows:—

(1) Removal of the cortex of one cerebellar hemisphere produces no degeneration of the fibres in the spinal cord.

(2) Degeneration in the spinal cord is directly proportionate to injury of the vestibular nuclei, *i.e.*, Deiters' and Bechterew's.

(3) The cerebellar nuclei, *i.e.*, nucleus dentatus and nucleus fastigii (vel tecti), send fibres to the vestibular nuclei.

(4) The cortex of the hemispheres (*sic.*, *query* lateral lobes) appears to be the principal source of fibres passing from the cerebellum to the pons. Of all the fibres in the middle peduncle, however, but few relatively are cerebello-pontine; the majority are ponto-cerebellar.

(5) The cortex is directly connected with the nucleus tecti and the nucleus dentatus.

With all these conclusions our own results are in complete accord. But Thomas is further of opinion that some fibres leave the cerebellar cortex and pass by means of the restiform body to the nucleus lateralis and nucleus cuneatus of the bulb. He also leaves it undecided whether fibres do pass from the cortex to the superior cerebellar peduncle. The first of these points is contradicted by our experiments, and the second we are able to decide in the negative.

(VII.) *Ramon y Cajal* (1901).—This investigator has suggested that some fibres may pass directly from the cortex to the superior peduncle. His definite conclusions, as given in his text-book, however, are as follows:—

(1) The axones of the Purkinje corpuscles of the cortex terminate in the nucleus dentatus and the nucleus tecti.

(2) The axones of some of the cells of the nucleus tecti pass down the spinal cord to come into connection with the cells of the anterior cornua.

(3) The axones of the cells from the dentate nucleus bifurcate, one limb entering the superior peduncle, the other passing down the lateral region of the bulb and spinal cord, but with indefinite termination.

In other parts of his work he apparently accepts the position put forward by Marchi, and it is to be observed that he always specially speaks of the descending spinal cord fibres as the "*via descendente de Marchi.*"

He also gives a figure of descending degeneration in the spinal cord of a guinea-pig after extirpation of one cerebellar hemisphere, but it is obvious from the other facts of the experiment that the lesion was a very extensive one.

(VIII.) *Probst* (1902).—Since the work of the investigators hitherto cited a systematic research on the afferent and efferent tracts of the cerebellum has been published by Probst. His plan was the same as ours, namely, to make lesions with aseptic precautions relying entirely on the Marchi method to follow the degenerated fibres. He does not anywhere describe that the lesions were absolutely restricted to the cortex of the cerebellum, and, in fact, all seem to have been combined with lesions of the cerebellar nuclei in varying degree of intensity. Although in our opinion it is not justifiable to argue that inasmuch as the number of fibres issuing in the cerebellar efferent paths is apparently proportional to the amount of the injury that the nuclei sustains, and therefore that no fibres pass from the cortex to the peduncles directly Probst considered that he was able to formulate the following conclusions :—

(1) That no fibres go from the cerebellar cortex into the spinal cord or into the thalamus.

(2) All the centrifugal fibres of the cerebellar cortex pass to the dentate nucleus or the roof nucleus or to Deiter's nucleus.

(3) Direct fibres from the cerebellar cortex can be traced to the pontine nucleus.

(4) Direct fibres pass from the cortex in the outer segment of the restiform body to the nucleus of the lateral columns.

(3) Anatomical Introduction.

(1) *Cerebellum of the Cat.*

The nomenclature of the different parts of the cortex of the cat's cerebellum is unfortunately at the present time under revision.[1] We therefore offer the following description based in the main upon the work of Elliot Smith. While we think it undesirable to employ terms which convey homologies that have not yet been accurately determined, certain names in common use will be found convenient. Thus, the cerebellum for physiological, and in part also for anatomical, reasons, has been divided into a mesial portion or *vermis*, and, on either side of this, a *lateral lobe*, which on the posterior surface of the organ is separated from the vermis by the *sulcus paramedianus*. These names we intend to retain.

The vermis.—This may best be divided, as Elliot Smith proposes, into three lobes—*anterior*, *middle*, and *posterior* (see fig. 1). The delimiting sulcus between the first two is now generally termed the *primary fissure*. This is a deep vertical cleft which is well seen in a mesial section of the whole organ. The cortex in front of this fissure may be called the *anterior lobe*, provided it is fully understood that it is separated on each side from the *lateral lobe* by the *primary fissure* turning downwards and forwards until it arrives at the surface of the *superior peduncle*.

(I.) The *anterior lobe* consists of some twenty-one folia which have been divided from below upwards into: (1) The *lingula*, consisting of one, or at the utmost three, folia. (2) The *lobus centralis*. This is composed of about six folia, and extends to the upper surface of the *inter-collicular commissure*. (3) The remainder of the *anterior lobe* is made up of about twelve folia, and has usually been described as the *culmen*, by which term we shall speak of it.

For the sake of simplicity we will next dispose of:—

(II.) The *posterior lobe*, which is formed by only two lobules, namely, the *nodulus* and the *uvula*, and is limited

[1] This is not the place to refer to the very important works on this subject by Bolk, Charnock Bradley and Elliot Smith; we can only refer the reader to their papers.

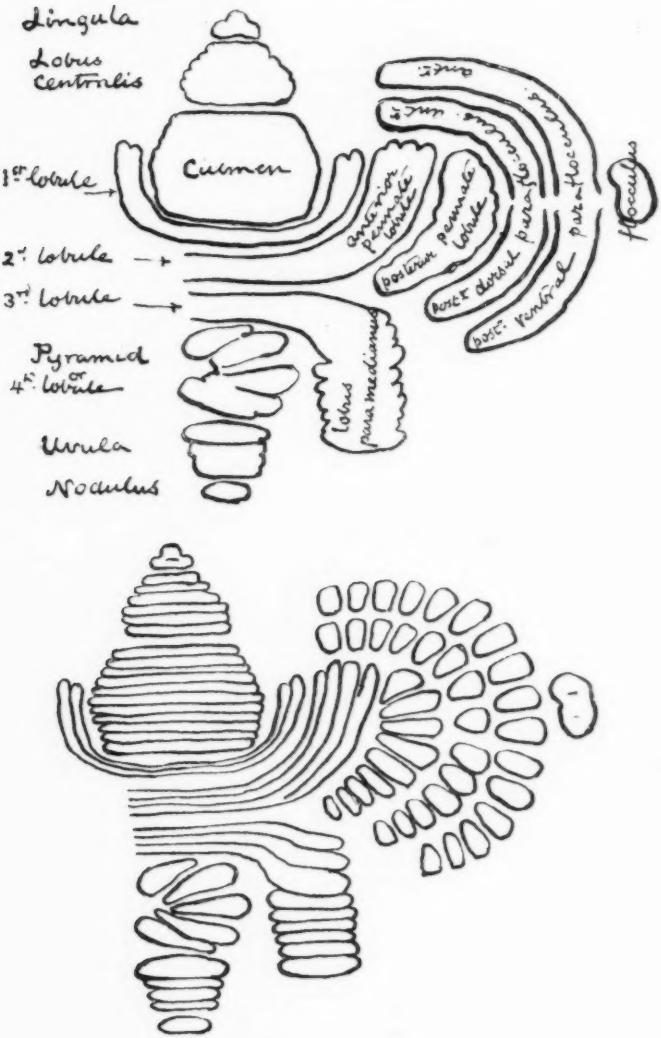
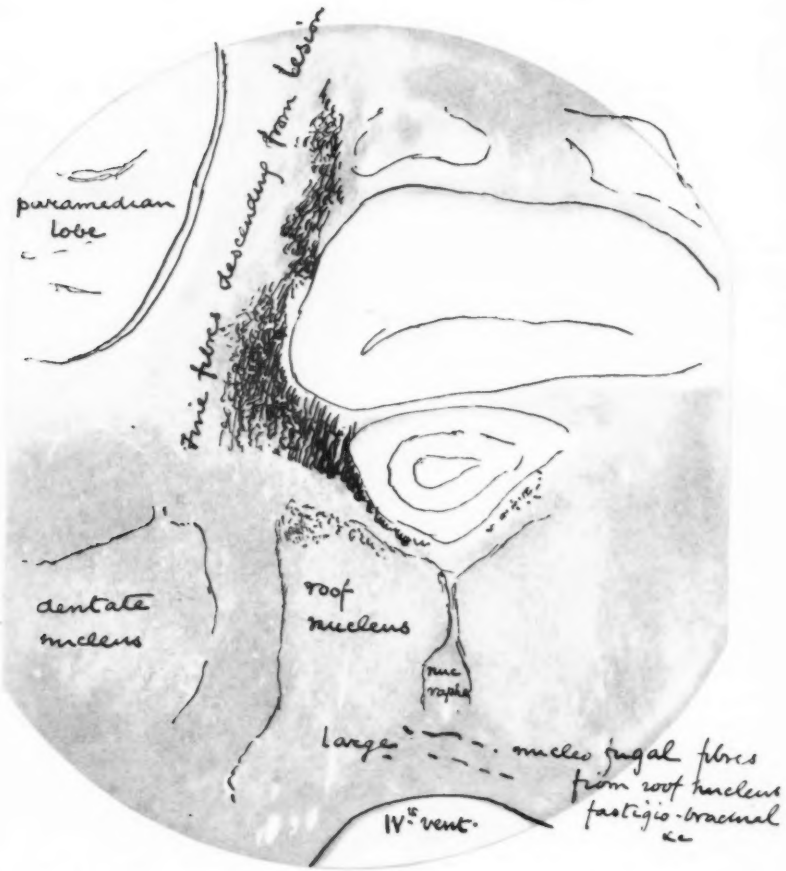


FIG. 1.

Cerebellum of cat. The cortex of the vermis and lateral lobes represented on the flat.

Fig 2. Exp^{t} 7 Cat: Oblique Frontal Section

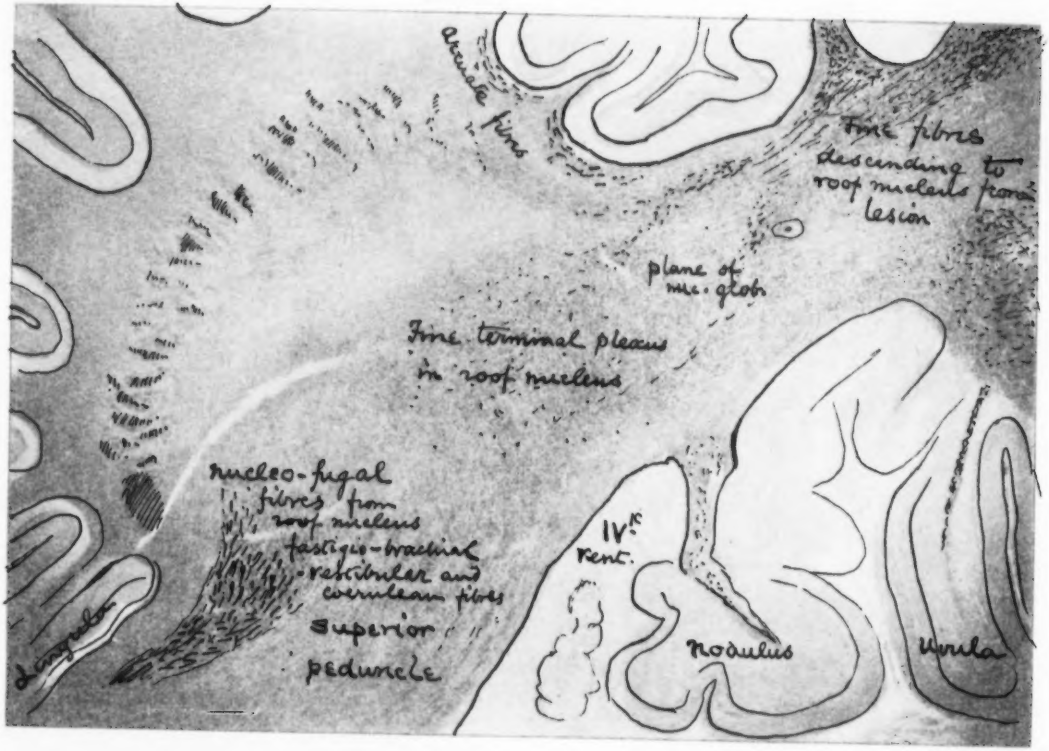


The dark shadow round the nucleus dentatus and between it and the nucleus fastigii is not due to degeneration, but only to the osmic reaction with the thick felt-like perinuclear plexus.

The dark shadow round the nucleus dentatus and between it and the nucleus fastigii is not due to degeneration, but only to the osmic reaction with the thick felt-like perinuclear plexus.

Cerebellum of cat. The cortex of the vermis and lateral lobes represented on the flat.

Fig. 3. Exp^{t} 6. Cat. Sagittal Section.



dorsally by a deep sulcus, the *secondary fissure* of Elliot Smith. The *nodulus* consists of only one folium which often has a double head. The *uvula* contains from four to five folia, the most dorsal of which lies on the *obex* of the *calamus scriptorius*, is always a large folium, and is bounded on its dorsal surface by the *secondary fissure*.

(III.) The *middle lobe* has been divided by Elliot Smith into four parts.[1] The (*a*) *first lobule* consists on the average of two folia which, situated like a horseshoe behind the anterior lobe, form the posterior lip of the primary fissure. The (*b*) *second lobule* is composed in the middle line of two or three folia, which spread out laterally into a series of folia arranged bi-pennately on the supero-anterior aspect of the *lateral lobe*. The anterior series of these pennate folia are usually from four to five in number, their long axes are antero-posterior, and they form a very definite quadrangular topographical feature of the roof of the *lateral lobe*. We shall speak of them collectively as the *anterior pennate lobule*. From the outermost of these five antero-posterior folia a series of about eight folia form the tip of the *lateral lobe*, and are continued posteriorly behind the antero-posterior folia before mentioned, so as to complete, though somewhat asymmetrically, the bi-pennate arrangement of the roof of the *lateral lobe*. They may be termed the *posterior pennate lobule*. Inasmuch as the side of the *lateral lobe* on its outer aspect is built up, as will be seen directly, of the *floccular lobes*, the bi-pennate arrangement of the roof together with the *paraflocculus*, has not inappropriately been termed by Elliot Smith the *supraflocular mass*. We have above suggested that this bi-pennate roof of the lateral lobe should be divided into a *pars anterior* (the anterior pennate lobule) and a *pars posterior* (the posterior pennate lobule). (*c*) The *third lobule*, which forms in the middle line the upper horizontal limb of the convoluted posterior *vermis*, includes from two to three folia, and extends outwards on each side into two vertical lobes forming the outer boundary of the *paramedian fissure*, and, therefore,

[1] Some French writers speak of the whole *vermis* as the middle lobe of the cerebellum.

properly termed the *paramedian lobes*; each of these is made up of from nine to ten folia. In contact with the outer surface of this vertical lobe lies the posterior border of the *paraflocculer mass*. (*d*) The *fourth lobule* is formed by the *pyramid*, and, in the cat, includes from four to five folia placed obliquely since it enters into the sigmoid convolution of the posterior vermis forming its lower horizontal branch.

We will now describe the remainder of the *lateral lobe* (or hemisphere of human anatomy), *i.e.*, the ventral two-thirds of the depth of the anterior, external and posterior aspects of the outer part of the lobe.

On the external or lateral aspect the folia are apparently arranged in three tiers, but on the anterior and posterior surfaces in only two. The lowest tier on the external aspect is made up anteriorly of the sessile *flocculus* and posteriorly by the *tuberculum acusticum*. The remaining two tiers extend horizontally round the lobe both forwards and backward, and are termed the *dorsal* and *ventral paraflocculus*. A portion of the *ventral paraflocculus*, where the anterior and exterior surfaces meet, is embedded in a special fossa of the *petrous bone*, and is spoken of as the *petrosal lobule*. Each tier of the *paraflocculus* is composed of from fourteen to sixteen folia.

These terms by which the various areas of cortex thus marked off may conveniently be known must, in the present state of our knowledge, be merely provisional until the exact homologies are ascertained. We therefore furnish diagrams of the lesion in each experiment, and photographs where these have been specially taken. The precise area of the lesion, however, is seldom well shown in a photograph of the actual specimen as when the dura mater is adherent to the neighbourhood of the wound the surface is necessarily ragged after dissection, and, on the other hand, undermined folia often appear normal (*cf.* plate IV.).

(4) Results.

We have thought it better to group our results in tabular form (Table I.). The columns of the table sufficiently ex-

plain themselves, but it will be understood that where the definite statement "*nil*" is made it means that there were no degenerated fibres to be found in that part after repeated re-examination of the various series of sections. It will be seen that there is a very close concordance of results obtained from lesions of the same district of the cerebellar cortex. We think that the explanation of this is to be found in the very simple plan on which the cerebellum is constructed and the homogeneity of its structure throughout.

(5) Summary.

(*a*) *Relation of the Cortex Cerebelli to the Cerebellar Peduncles.*

We believe that the results of our experiments embodied in the foregoing table justify the following general conclusions on the direct question whether fibres (presumably axones of the Purkinje corpuscles) pass directly into the cerebellar peduncles or not.

(1) No fibres issuing from the cortex cerebelli enter any of the peduncles.

(2) All fibres leaving the cerebellum by way of the peduncles have origin in one or other of the cerebellar nuclei.

(*b*) *Relation of the Cortex Cerebelli to the Nuclei Cerebelli.*

Our observations show that any given area of the cortex is in relation with a homolateral nucleus or nuclei. We have not found satisfactory evidence that the cortico-nuclear fibres cross the mesial plane.

This simple relation of the cortex and nuclei is illustrated in fig. 2, where, from a lesion in the vermis, fine fibres are seen streaming forwards to reach the dorsum of the roof nucleus.

The details of these relations are perhaps best shown in parallel columns as follows :—

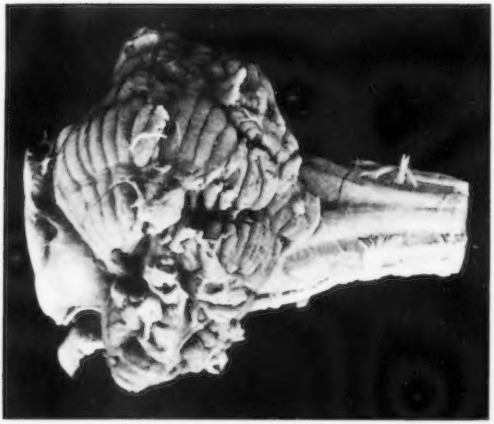
TABLE II.

Portion of Cortex Cerebelli.	*Nucleus with which it is in relation.*[1]
(1) Anterior pennate lobule.	Nucleus fastigii. Nucleus dentatus.
(2) Posterior pennate lobule.	Nucleus dentatus.
(3) Flocculus, paraflocculus.	Nucleus fastigii.
(4) Anterior pennate lobule. Anterior Paraflocular lobule. Paramedian lobe.	Nucleus dentatus. Nucleus globosus. Nucleus fastigii. Nucleus vestibuli.[2]
(5) Omitted for involvement of nucleus fastigii.	
(6) Culmen (five posterior folia).	Nucleus fastigii. Nucleus globosus.
(7) Culmen and all middle lobe.	Nucleus fastigii. Nucleus dentatus. Nucleus vestibuli.[2] Nucleus globosus.
(8) Left half of lobus centralis, culmen and left four-fifths of first and second lobule of middle lobe.	Nucleus dentatus. Nucleus fastigii. Nucleus globosus. Nucleus vestibuli.[2]
(9) Second lobule of middle lobe and anterior pennate lobule.	Nucleus dentatus. Nucleus fastigii. Nucleus globosus. Nucleus vestibuli.[2]
(10) Three folia of third lobule of middle lobe	Nucleus fastigii (very few fibres).
(11) Third and fourth lobules of middle lobe.	Nucleus fastigii. Nucleus globosus. Nucleus dentatus.
(12) Fourth lobule of middle lobe (pyramid).	Nucleus fastigii.
(13) Fourth lobule of middle lobe and two outer folia of uvula.	Nucleus fastigii.
(14) Lowest folium of fourth lobule of middle lobe and two folia of uvula.	Nucleus fastigii.
(15) Whole uvula.	Nucleus fastigii. Nucleus globosus.
(16) Ventral half of fourth lobule of middle lobe, uvula, and two anterior folia of first lobule of middle lobe.	Nucleus fastigii. Nucleus globosus.
(23) Lobus quadrangularis and side of culmen.	Nucleus fastigii. Nucleus globosus. Nucleus dentatus.

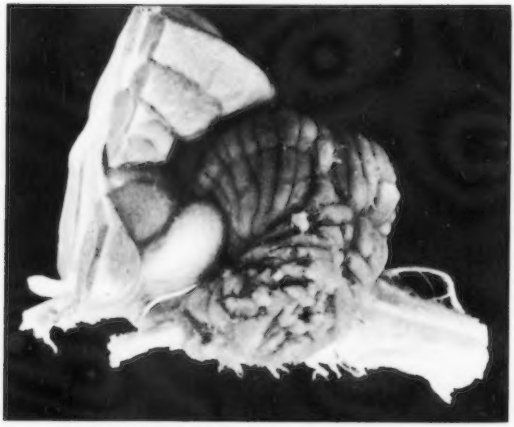
[1] Where more than one is concerned the nuclei are arranged in the order of more extensive anatomical connection.

[2] It will be observed that the association of the nuclei vestibuli has only been noted in extensive lesions. It is conceivable that secondary circulatory changes may possibly account for the more extensive association.

PLATE I.



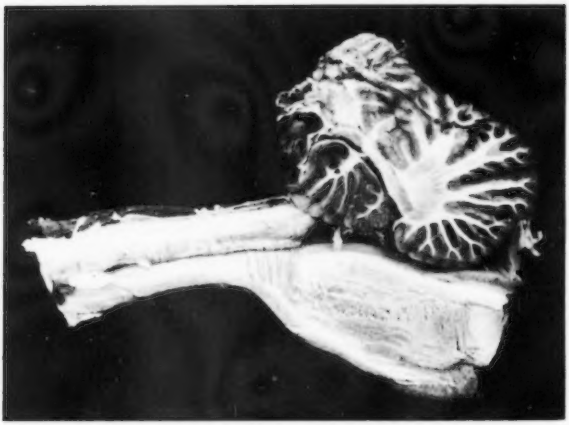
EXPERIMENT No. 1.



EXPERIMENT No. 2.

The numbers refer to the Experiments given in Table I.

PLATE II.



EXPERIMENT No. 5.

EXPERIMENT No. 6.

The numbers refer to the Experiments given in Table I.

From the foregoing it is evident that the *roof nucleus*, nucleus fastigii (see fig. 2), is of chief importance among the nuclei, for it is in relation practically with all parts of the cerebellar cortex, and must consequently possess a very important function in associating the cortico-fugal impulses.

Further, it has for long been known to have special association with the vestibular nuclei (Kölliker, Edinger, Ramon y Cajal). In our preparations, where very limited lesions of the nucleus fastigii had been made, it has been quite easy to follow both coarse and medium fibres over the superior cerebellar peduncle to the gray matter surrounding that structure, and to the locus cœruleus (see fig. 3). We would suggest the terms "fastigio-brachial" and "fastigio-cœrulean" to denote these fibres, but their full discussion and description must be reserved for another communication.

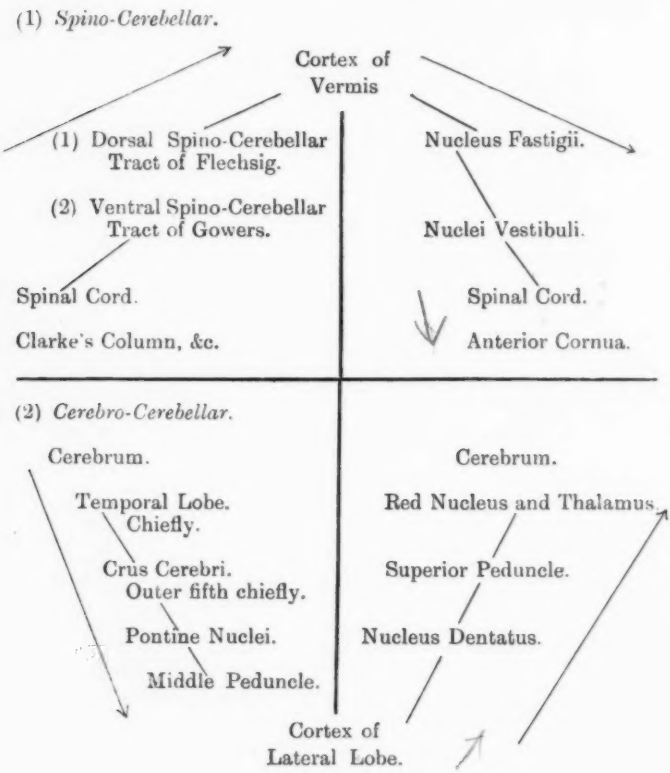
The *nucleus globosus* has no relation with the lateral lobe or flocculus. It is essentially a nucleus appertaining to the vermis system, and its connections extend as far laterally as the paramedian lobe.

The *nucleus dentatus* is intimately associated with the pennate lobules, but not with the flocculus and paraflocculus. These two important portions of the cortex are connected with the roof nucleus, which fact fully confirms the anatomical conclusions of Elliot Smith as regards the relation of the floccular region to the postero-inferior part of the vermis.

As regards any association of the nucleus dentatus with the mesial mass of the cortex or vermis, our experiments suggest clearly that, first, there is no relationship between the nucleus dentatus and the anterior or posterior lobes, *i.e.*, lingula, lobus centralis, culmen, uvula, and nodulus; and, secondly, that the only connection between the nucleus dentatus and the vermis is with the middle lobe of the latter. Finally, of the middle lobe our evidence goes to show that it is only the three anterior lobules which are connected with the dentate nucleus, and that the fibres making such connection are relatively few.

The intrinsic arrangement of the cerebellar cortex and nuclei can be therefore regarded as divisible into two dis-

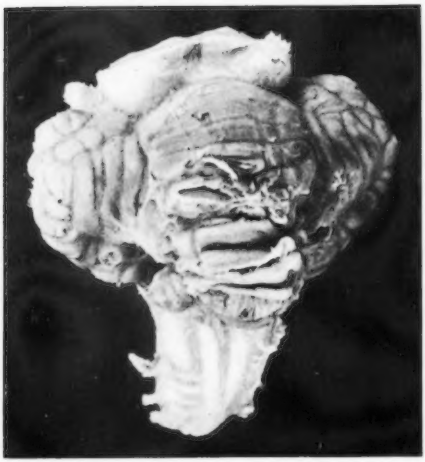
tinct organs, a spino-cerebellar and a cerebro-cerebellar looped system, of which the parts are thus arranged:—



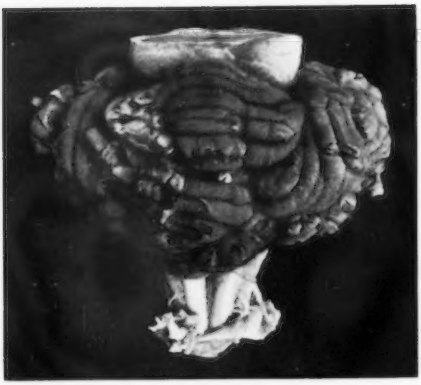
In considering the relation of the cerebellum to the cerebral pallium, much stress has been laid in the past on the excitable or so-called motor area of the cortex cerebri as being probably that part of the pallium which is definitely associated with the pontine nuclei and so with the cerebellum.

That this association exists is amply established by the investigations of Luciani, Ramon y Cajal, Simpson,

PLATE III.



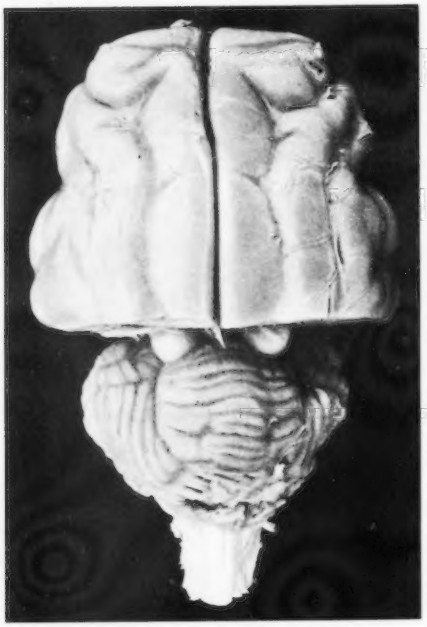
EXPERIMENT No. 7.



EXPERIMENT No. 8.

The numbers refer to the Experiments given in Table I.

PLATE IV.



EXPERIMENT No. 9.

EXPERIMENT No. 10.

The numbers refer to the Experiments given in Table I.

Beevor and Horsley, proving the existence of an extensive system of intrapontine pyramidal collaterals.

It appears to us, however, that this point requires further consideration, and, as we have in the above scheme put the temporal region as being that part of the cortex cerebri which is most directly connected with the cerebellum, we must now state the grounds for this view.

Taking first the crural tracts, the opinion of Dejerine is, of course, universally recognised to be correct, viz., that the temporo-pontine bundle occupying the outer fifth of the cerebral peduncle finds origin in the middle third of the temporal lobe and terminates in the pons (frontal segment according to unpublished experiments by Beevor and Horsley). Now, inasmuch as the temporal pallium is the seat of projection of the auditory paths, it is not unreasonable to regard it, rather than the excito-motor gyri, as being also the area in which the vestibular sensations are represented.

This view of the pallial vestibular representation has also received the authoritative support of Chas. K. Mills, who, in his most recent diagram (*Medical News*, November 5, 1904), figures "equilibratory" and "orientation" in the posterior third of the temporal lobe (second and third gyri).

Since the ponto-cerebellar fibres cross the raphe, it follows that the temporal pallium of one side would correspond with the opposite lateral lobe of the cerebellum, which is well known to be the case, as far, at any rate, as the whole cerebral hemisphere is concerned.

Further, the records of cases of defective development of the cerebellum hitherto published (*cf.* Risien Russell, Herringham, Lewis, &c.) show that the part of the cortex cerebelli to fail first is the lateral lobe, or, in other words, that part which, having cerebro-pallial relations, is necessarily later in evolution and succumbs, therefore, more readily to adverse circumstances.

The solution of this question will afford the basis of a rational explanation of the phenomenon of cerebellar circumduction, or "*mouvement de manège*," but at the present time it is uncertain to what degree "*mouvement de manège*"

is due in cases of cerebral lesion to the direct influence of the cerebrum or to distal action (by pressure, &c.) of the cerebellum itself.

At least this we suggest is true, that of all the regions of the cerebral cortex which send either direct axones or collaterals to the pontine nucleus the temporal region is the chief.

(*c*) *The Arcuate System.*

The arcuate fibres which connect any given folium with its neighbour are of obvious importance in an organ the structure of which is so remarkably uniform throughout. We have, as far as possible, noted in Table I. in each experiment any special points respecting the arrangement of the arcuate fibres which required mention, and, therefore, now only offer the following general conclusions on this matter.

(1) Very few arcuate fibres pass from the vermis to the lateral lobes, and then never beyond the plane of the paramedian lobe.

(2) Arcuate fibres pass, as a rule, laterally to only the second folium from the edge of the lesion, and very exceptionally as far as the third folium.

(3) Extensive lesions of the vermis, especially of the middle lobe, are followed by widespread degeneration of arcuate fibres having an antero-posterior course.

(4) Extremely few arcuate fibres pass to the nodulus.

The doctrine of the independence of the vermis and lateral lobes respectively is thus also confirmed by study of the arcuate system of communicating fibres.

(*d*) *Size of the Various Intrinsic Fibres of the Cerebellum.*

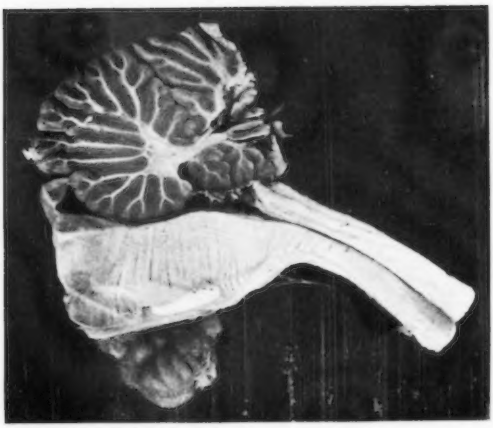
The following list shows the general size of the different groups of intrinsic fibres of the cerebellum :—

(1) Cortico-nuclear : Medium and fine.

(2) Arcuate (intercortical) : Fine.

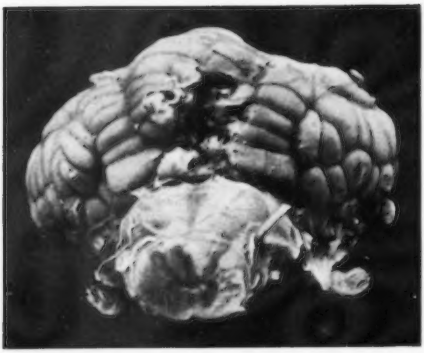
PLATE V.

EXPERIMENT No. 12.

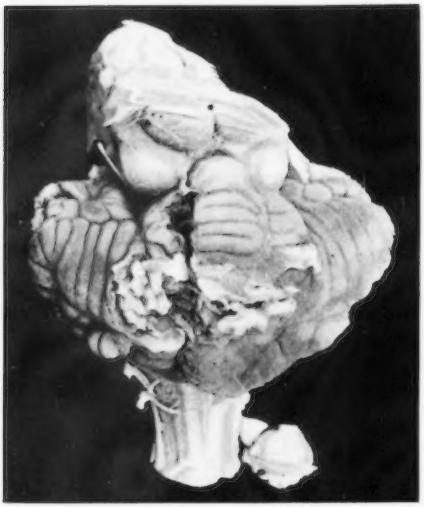


EXPERIMENT No. 16.

The numbers refer to the Experiments given in Table I.

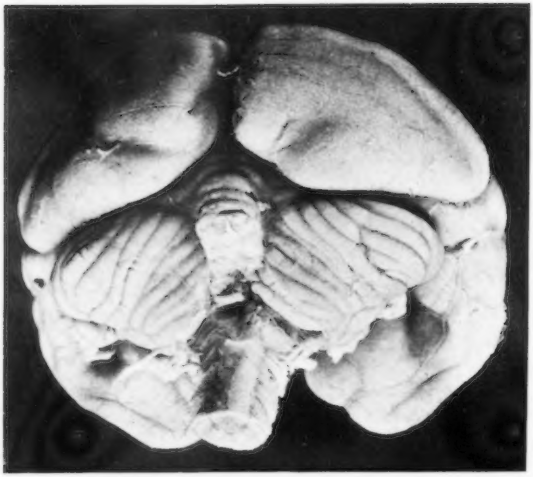


EXPERIMENT No. 17.

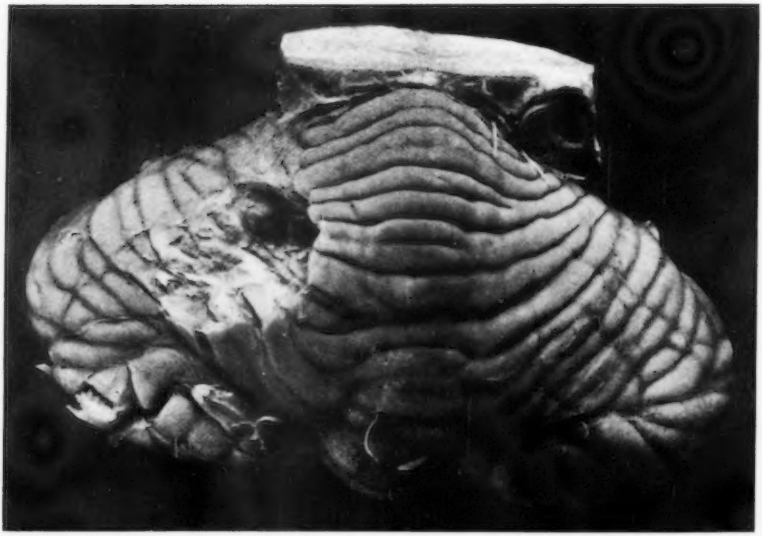


EXPERIMENT No. 18.

The numbers refer to the Experiments given in Table I.



EXPERIMENT No. 22.



EXPERIMENT No. 23.

The numbers refer to the Experiments given in Table I.

(3) Nucleo-peduncular: Superior, large; middle, fine; inferior, medium.

The differences in size of the various groups are well shown in fig. 3, which is a photograph of a section showing the cortico-nuclear and nucleo-fugal fibres in their relative proportions in the cat.

References (pp. 16-18).

(I.) "Sull' origine e. decorso dei peduncoli cerebellari, &c," *Firenze*, 1891, and *Riv. Sper. d. Fren. s. Med. Leg.*, 1886-1891.
(II.) *Philosophical Transactions of the Royal Society*, 1894.
(III.) *Philosophical Transactions of the Royal Society*, 1896.
(IV.) *Neurologisches Centralblatt*, 1895, p. 434.
(V.) *Prager Medizinische Wochenschrift*, 1895, No. 14.
(VI.) *Le Cervelet*, Paris, 1897.
(VII.) "Textura del Sistema Nervioso, &c." Madrid, 1899-1902.
(VIII.) *Archiv. f. Pschiatrie*, xxxv., p. 692.

Description of Plates.

The plates hereto appended are the actual photographs taken of the cerebellum in most of the cases included in Table I.

Reference must be made to Table I. for the accurate delimitation of the lesion especially where this has had an undermining effect.

DIFFUSE SARCOMATOUS INFILTRATION OF THE SPINAL PIA MATER.

BY STANLEY BARNES, M.D., BSc., M.R.C.P.,
Pathologist and Medical Registrar to the Queen's Hospital, Birmingham.

ALTHOUGH many cases of multiple tumours of the central nervous system have been published, yet the means whereby these tumours are spread are still very imperfectly understood. If we are to assume that in the matter of new growths the central nervous system is analogous to the other parts of the body, we are driven to adopt the hypothesis that, as a general rule, a new growth there is primary in only one situation, and that in any cases of multiple growths one of the lesions was primary and the others were secondary—*i.e.*, are the result of metastatic "infection." It therefore becomes a matter of considerable interest to ascertain whether the means of dissemination can be explained adequately in the present state of our knowledge in the various cases now published in the literature.

Two cases recently occurred at the Queen's Hospital, Birmingham, which throw considerable light on this subject, and, in recording the clinical and *post-mortem* phenomena, I have ventured also to append an abstract of certain similar cases to which I have had access. I am indebted to the kindness of Dr. Carter for permission to publish these cases.

Case 1 (Barnes).—On January 3, 1903, a boy, 13 years of age, was admitted to the Queen's Hospital complaining of progressive loss of eyesight for two years.

Family history.—Father alive and well, has never had syphilis. Mother died at 36, of puerperal fever. There are five brothers and one sister, all well; there have been no other children, or mis-

carriages. There is no suggestion of syphilis in any of the other children.

Previous history.—Always strong and healthy.

Present illness.—Began two years ago with attacks of morning vomiting, having no relation to food, and with progressive loss of sight About the same time he also complained of occasional headaches, which are stated to have been of only moderate severity. He also became generally weak and wasted, but after an illness lasting about six weeks, he began to improve—the sight improved sufficiently to enable him to read large print, the vomiting ceased, and the headache disappeared. Two months ago the symptoms recurred: morning vomiting, further loss of sight and frontal headache being the prominent symptoms.

State on admission.—A pale, thin boy of 13, lay comfortably in bed. Temperature, 97·5; pulse, 96; respirations, 24.

Nervous system.—Intelligence, memory and speech normal. Smell good on both sides.

Vision was reduced to perception of light only, on both sides. The optic discs both showed optic neuritis with early atrophy.

There was doubtful weakness of the left external rectus muscle, but no obvious squint. The pupils were equal and dilated; they did not react to light.

Taste slow, but otherwise normal. Fifth nerve normal. There was some supranuclear paralysis of the right side of the face. Hearing normal on both sides. 9th, 10th, 11th, and 12th nerves normal.

Motor system.—There was no local wasting or paralysis other than in the face. General muscular wasting considerable. Sensation normal throughout. Co-ordination good. Gait natural for a blind boy.

Reflexes.—Organic, normal. Plantars were lively, with flexor response. Knee-jerks were absent. Abdominal and cremasteric equal and well marked. No trophic disturbances.

The other systems, circulatory, respiratory, &c., were examined and found normal.

Progress whilst under observation.—On January 5 he had an attack of cramp affecting both hands simultaneously and lasting about five minutes; the hands were flexed upon the forearm, the fingers being extended; there was no twitching, and consciousness was not lost.

On January 8, a convulsion occurred. It began with a loud scream, and then at once the back was arched and the legs and arms were extended; the wrists were flexed and the fingers

extended; he was unconscious for a few seconds. This was followed a few minutes later by a second convulsion; it was of the same type but was followed by clonic spasms of the left side of the face, of the arm and leg. He was drowsy afterwards, and remembered nothing of the attack.

On January 11, it was noted that the respiration was irregular, being somewhat of the Cheyne-Stokes' type. He was evidently very much worse, and in the night moaned continually in his sleep. Next morning he was very sick, complained of feeling ill, and soon lapsed into a semi-comatous state. The toes of the left foot were flexed, and then spasmodic contractions occurred in the left leg for a few minutes; then gradually the arms were extended and the fingers and thumbs twitched. The left side of the face was also convulsed, and then the back arched, the head was thrown back in tonic spasms for a few moments, and then he gradually relaxed and the fit was over. The time occupied by these movements was about fifteen minutes. He gradually became conscious, but remained very dull.

Next day (January 13) he could be roused and replied sensibly to questions, but tended to drop asleep whilst one was talking to him. Breathing was still very irregular.

On January 17 he was more comfortable, and the headache and sickness had passed off. No more convulsions had occurred. Both external recti seemed partly paralysed, and vision was now absent.

On January 21, he began to pass urine and fæces into the bed; he was now wasting considerably.

From this date forwards he went slowly down hill; he became more and more drowsy, having occasional convulsions of no great severity, and of the type already described. By June 1, he was so wasted that despite the most careful nursing bed-sores occurred on the hips and sacrum; there seemed to be no subcutaneous fat left, and the muscles were reduced to about one-third of their original size. There was now no rigidity, and for several weeks all the reflexes (including the plantar) had been absent.

He died after a short convulsion on June 21, 1903. For a week before death, the temperature had not risen above 95·4°. During his stay in hospital, it never rose above 99°.

He was treated with iodide of potassium for a long time; for the headache, potassium bromide was usually given.

Post-mortem Examination.

The autopsy was performed on June 22, twenty-four hours after death.

The body was greatly emaciated, and rigor mortis was general.

The thoracic and abdominal viscera were carefully examined and found normal. There was no sign of growth, nor was anything found suggestive of tuberculosis or of syphilis.

The head was unusually large, especially in the bi-parietal diameter. The skull was of natural thickness. The dura mater was not thickened, and was everywhere free from adhesions to the arachnoid.

The convolutions of the brain were of natural size and arrangement, and were considerably flattened out. The sulci contained very little fluid.

The pia and arachnoid over the vertex were normal except in the left frontal region; here was a somewhat circular patch about the size of a crown piece, which in the fresh state was of an opaque greenish-white colour, and at first sight appeared to be an island of purulent lepto-meningitis. The arachnoid covering this patch was smooth and not adherent to the dura mater.

At the base of the brain the dura was normal, but the other membranes were involved in a massive infiltration of solid tissue. This thickening of the pia-arachnoid extended as far forwards as the optic chiasma and anterior perforated spaces, and laterally to the apices of the temporo-sphenoidal lobes. All the natural hollows of the base of the brain were more or less completely filled up with this material, and the upper and lower boundaries of the pons were obscured on the surface. The meninges on the under surface of the cerebellum were a little thickened, but at the region where the medulla strikes free from the cerebellum the thickening, though still most marked on the under surface, almost surrounded the spinal axis.

The laminæ were removed and found normal. The theca looked normal externally, and when opened was found to be of natural thickness, and not adherent unnaturally to the subjacent membranes. It was now seen that the cord was almost entirely surrounded by the thickening of the membranes which extended throughout its entire length (see fig. 1). The surface appearance was like that of the material at the base of the brain; of an opaque pinkish colour, it presented many nodosities over its coarsely uneven surface. In the main there was more growth over the posterior than over the anterior regions; the thickness varied up to a maximum of twelve millimetres.

The emerging nerves, cranial and spinal, with the exception of the first pair of cranial nerves, all passed through the thickened

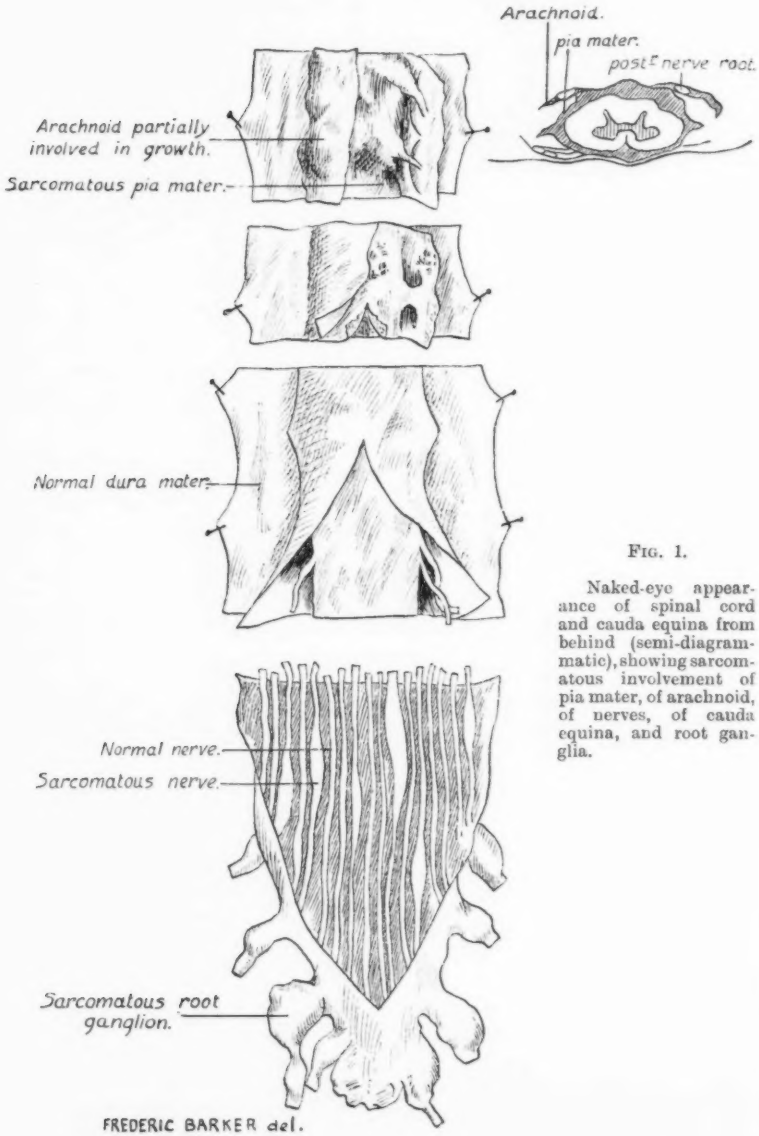


FIG. 1.

Naked-eye appearance of spinal cord and cauda equina from behind (semi-diagrammatic), showing sarcomatous involvement of pia mater, of arachnoid, of nerves, of cauda equina, and root ganglia.

membranes. The nerve roots were not markedly atrophic. The posterior root ganglia throughout the spinal region were enormously enlarged, those of the cauda equina being about the size of horse-beans; in the cervical region they were somewhat smaller, but the Gasserian ganglia were as large as those of the cauda equina. A few similar discrete nodes were present along the intra-thecal course of the nerves forming the cauda equina.

Many of the peripheral nerves were examined, but nowhere outside the intra-spinal or -cranial course could any thickening be discovered.

A series of horizontal sections was then made through the hardened brain. At the level of the internal capsule, it was found that a large tumour was present in the left frontal region. The tumour as a whole was reddish-grey in colour, with some streaks of yellow near its centre and hinder end; it was about the size of a Tangerine orange, and roughly triangular in shape, the apex being directed backwards and inwards and lying near the foramen of Monroe. The hinder and inner end of this tumour had broken down, clearly rupturing into the lateral ventricle; at this point the tumour was hollowed out and had a rough and uneven base facing the ventricle, and the deeper yellow colour showed that some time previously an extravasation of blood had occurred here.

The growth extended forwards in the white matter from this point as far as the gray matter of the frontal cortex; in one place the tumour had grown along the course of the entering cortical vessels, and then involving the subpial tissues had formed the greenish-grey mass on the surface which had previously been mistaken for inflammatory exudate. Except at this point, the tumour was sharply bounded from the brain tissue, which seemed to be pushed in front of it rather than invaded by it. Behind, the caudate nucleus was displaced about an inch backwards, but was not invaded by the growth.

Running sagittally forwards from the hinder end of the tumour was a yellow line indicating an almost complete antero-posterior fissure in the growth. On opening out the tumour at this point, it was obvious that this cleft represented the remains of the anterior cornu of the lateral ventricle which had been almost obliterated by the tumour which had been growing on both sides of it.

It therefore seemed clear that this tumour had its origin in the ependyma lining the anterior cornu of the ventricle; that it

had grown forwards, pushing the white matter in front of itself and causing it to atrophy without infiltrating it appreciably; that it had similarly pushed backwards the caudate nucleus; that its further growth had obliterated the cavity of the ventricle in this region, and that at some later period it had burst into the lateral ventricle near the left foramen of Monroe.

No other large growth was found in the brain, but in view of the peculiar arrangement of the tumour mass around the spinal cord the ventricles of the brain were further explored. It was then found that several gelatinous masses were present, growing from the membranous lining of the ventricles of the central nervous system. The largest of these was found in the dependent part of the posterior cornu of the left lateral ventricle; it was a sessile and rather villous growth, which projected about 5 mm. into the ventricle. The choroid plexus in the same region contained a mass of growth about 6 mm. in diameter, but was not adherent to the other growth.

Scattered throughout the third ventricle, the aqueduct of Sylvius and the fourth ventricle were smaller masses, often—at the base of the fourth ventricle—forming a soft continuous layer of growth; the pineal body and the velum interpositum were much thickened by tumour mass.

On more careful examination of the thickened membranes, it was seen that the pia mater was more extensively infiltrated than the arachnoid. Although the latter was in many places adherent to the pia mater, in others it was only normally attached and preserved its naturally delicate structure. In other regions, as in the upper dorsal region of the cord (see fig. 1), the arachnoid was involved and adherent to the pia mater posteriorly, but was free on the anterior surface.

Microscopic Examination.

The growth in the frontal lobe is richly cellular, with a slight basis of neuroglia. The cells are spindle shaped with large oval nuclei and a comparatively small amount of protoplasm; they are arranged for the most part in whorls, which occasionally have a blood-vessel for their centre. Scattered irregularly through the tumour are found in considerable numbers large deeply-staining multinuclear cells.

The growth in the choroid plexus of the lateral ventricle showed a basis of fibrous tissue arranged more or less regularly like the stroma of a scirrhous cancer, each space containin groups

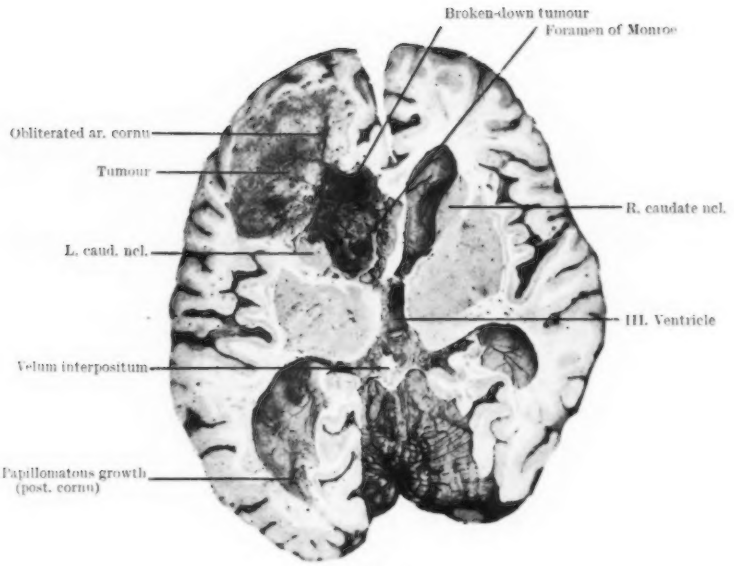


FIG. 2.

Horizontal section of brain of Case 1, showing the frontal tumour and point of rupture into foramen of Monroe.

of cells with comparatively large oval nuclei; the growth here is typical of alveolar sarcoma.

Around the cord the growth presents similar characteristics to those described above; the amount of fibrous tissue varies considerably in different parts, some being richly cellular and resembling the growth in the brain, whilst others are similar to that in the choroid plexus. The growth throughout is highly vascular. The emerging roots, particularly the posterior, are considerably invaded by the growth, which in many places has split up the rootlets into their constituent bundles, and has occasionally even separated the individual fibres from one another. Very few fibres seem to be destroyed altogether.

At the edge of the cord the growth is attached at short intervals by pial in growths; none of these seem to carry into the cord any of the special cells of the tumour. At the emergence of the rootlets, neuroglia fibres pass directly into the tumour, but are almost immediately lost in the fibrous stroma of the latter.

In the Gasserian ganglion, the growth is of the same character as in the choroid plexus. There is a large amount of fibrous tissue present, and the tumour cells are invading the ganglion along its interstitial tissue in such a way that there is a minimum of destruction with a maximum of infiltration. The posterior root ganglia show the same changes.

From several regions of the cord, sections were stained by the Nissl method, but no marked changes in the nerve cells were found. A few of the anterior horn cells showed early chromatolysis, and in some of them there is marked eccentricity of the nucleus, but none show more severe degeneration.

Stained by the Marchi method, many of the posterior roots showed considerable degeneration proximal to the root ganglia; the rather diffuse staining suggested that the degeneration was not all of the same age, but that some had been present for a considerable time.

The posterior columns showed marked ascending degeneration of a diffuse character, evidently derived from partial degeneration of most of the entering posterior roots. At the periphery of the cord, where the encircling tumour was attached, a few scattered degenerated fibres were seen; they did not appear to belong to any particular tract.

Stained by the Weigert-Pal method, there was well-marked diffuse degeneration of the columns of Goll and Burdach. All the posterior roots passing through the tumour seem to be partially degenerated, though not all to the same extent. The anterior roots are less affected.

Case 2.—H. H., aged 50, was admitted to the Queen's Hospital on February 1st, 1905, with pain in the abdomen. On admission, he was a well-developed man, and the symptoms he exhibited suggested early carcinoma of the stomach. He complained of no headache, but vomited occasionally, and there was no hydrochloric acid in the gastric juice after a test breakfast. An indefinite resistance in the epigastrium and an enlarged gland in the left supra-clavicular region were the only definite physical signs found. During the next few weeks he became curious in

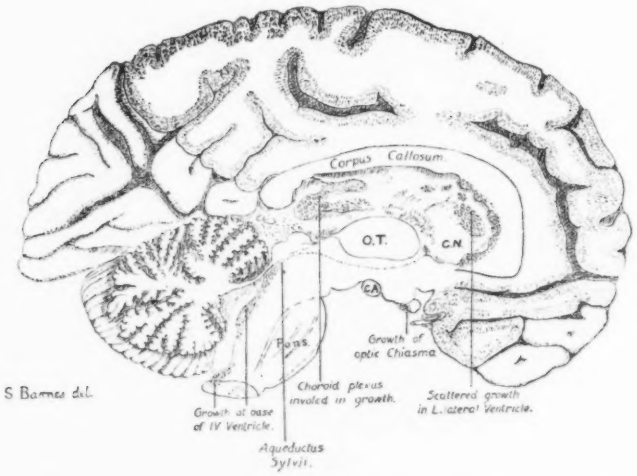


FIG. 5.

Case 2.—Sagittal section of brain, a little to the left of the middle line. Masses of growth are seen on the walls of the lateral and fourth ventricles, and the optic tract has become invaded by extension through the floor of the third ventricle. The growth is shaded with small circles.
C.N. cauda nucleus; O.T. optic thalamus; C.A. corp. albic.

manner with hesitating speech and more persistent vomiting, although this was never severe. He died on February 26, 1905.

At the autopsy, held the following day, there was found a large mass of retro-peritoneal growth which was adherent to but which had not perforated the duodenum. The iliac, aortic, posterior mediastinal and supra-clavicular lymphatic glands were all enormously enlarged, and on microscopic section gave the appearance of alveolar sarcoma or endothelioma.

FIG. 3.

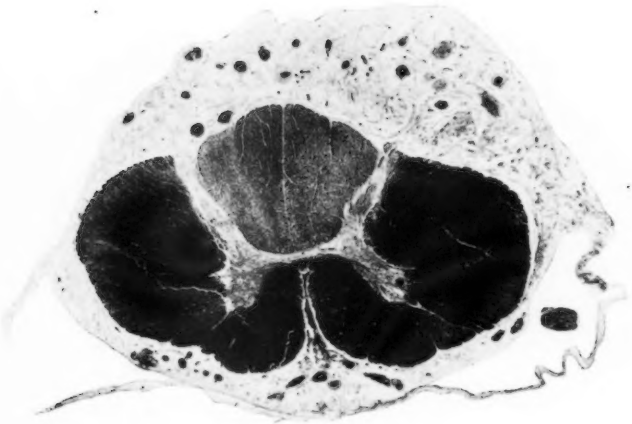


FIG. 4.

Photographs of sections of the spinal cord (Case 1), stained by the Weigert-Pal method. Degeneration of posterior columns and of anterior and posterior roots as they pass through the surrounding tumour. Arachnoid only partially involved.

The brain was then examined. On the right side the temporo-sphenoidal lobe contained a large diffusely infiltrating growth which invaded the tentorium cerebelli, and extended deeply so as to appear on the inner surface of the lateral ventricle near the foramen of Monroe. The surface of the growth facing the ventricle was extremely soft and ragged. On further inspection, it was found that the velum interpositum was also invaded by growth (by continuity), and that many discrete superficial masses of a similar nature were scattered over the surfaces of the lateral ventricles of both sides; the drawing (fig. 5) shows the inner aspect of the left lateral ventricle (sagittal section of the brain, just to the left of the middle line). The base of the aqueduct of Sylvius was free from growth, but the bases of the third and fourth ventricles contained several discrete masses tending in places to become confluent. At the base of the third ventricle in the middle line the lamina cinerea was infiltrated in its whole depth, and the growth had here invaded by extension the adjacent optic chiasma. Otherwise there was no sign of any involvement at the base of the brain.

The tentorial surface of the cerebellum on the right side immediately opposite the tumour in the temporo-sphenoidal lobe was covered with a thick milky adherent pia-arachnoid, which on section showed many cells indistinguishable from those of the growth, and which were not merely inflammatory exudation.

The spinal cord showed no abnormality, its membranes being normal throughout. Most of the posterior root ganglia were removed at the same time; all those of the cauda equina, and some of those of the dorsal region were found to be greatly enlarged—to about twice their normal size. The other ganglia of the cervical and dorsal regions were apparently normal. On macroscopic section of the enlarged ganglia the ganglion proper was seen to be intact, but the tissues around the point where the posterior root enters the ganglion were much matted and thickened.

Sections taken through the large growth in the brain showed masses of oat-shaped cells with very little stroma—a very simple type of growth, but evidently of great malignancy. The other masses on the lining of the ventricular wall showed a similar structure.

Several sections were taken through the spinal cord at various levels, but at no point could any definite meningeal growth be made out, the cord appearing when stained by the Van Gieson method to be normal.

Sections were then taken through the enlarged posterior root ganglia of the cauda equina. All of those examined showed well-marked aggregations of cells identical in form and size with those constituting the growth in the brain. These cells were arranged irregularly in groups, occupying the open lymphatic spaces immediately around the ganglia and the roots of the nerves, particularly the posterior, as they neared the ganglion from the cord. There was no sign that any of the nerve-fibres or cells were destroyed in any of the specimens examined. It was clear that there was an early infiltration of the root-ganglia by tumour cells, and there could be little doubt that their focus of origin had been the mass of growth in the cerebrum from which the metastases had been wafted by means of the cerebrospinal fluid.

Case 3 (Coupland and Pasteur).[1]—The patient was a single woman, 22 years of age. Her only previous illness had been rheumatic fever.

Her fatal illness began with bilious headaches and morning vomiting; pain in the shoulders and arms, and in the occipital region of the neck. Some delirium and motor weakness were soon added to her other symptoms, and it was then found that well-marked optic neuritis was present, and that both external recti were paralysed; the knee-jerks were lost. There were no convulsions.

Post mortem.—There was an excess of cerebrospinal fluid. The medulla and pons were invested with a jelly-like growth in which both sixth nerves were imbedded. The theca was adherent to the cord at one or two points. The cord was ensheathed in its whole length by a semi-transparent soft growth, which was most abundant on the posterior and lateral aspects. Many nodules were present on the intraspinal nerve-roots. Microscopically, the tumour was a small round-celled sarcoma.

Scattered growths were also found on the arachnoid at the base of the brain.

Case 4 (Coupland and Pasteur).[1]—After a fall down stairs, a girl, 4½ years old, became irritable, developed a squint, lost her power of walking, had pain in the head, and later optic neuritis. There was no anæsthesia or local paralysis. Difficulty in raising the eyelids, weakness of the trunk muscles and dilatation of the frontal veins followed. Before death, both arms were totally paralysed, but the feet never completely lost their power. There were no convulsions.

Post mortem.—There was increased intracranial pressure and excess of fluid in the ventricles. Beneath the arachnoid covering

the inferior surface of the cerebellum was a layer of greenish material which penetrated into the nervous tissue for about half an inch. The middle lobe of the cerebellum was almost entirely replaced by soft vascular growth. Few scattered islets of tumour formed thin plaques at the base of the brain in the pia mater. Over the whole cord the pia was thickened posteriorly, the maximum thickness being about 6 millimetres. In places there was growth on the lateral and posterior aspects of the cord.

Microscopically, the growth was a soft sarcoma.

Case 5 (Ollivier)[2].—A boy, 11 years of age, had complained of attacks of headache accompanied by retraction of the head and recurring every six months. There was vomiting, pain in all four extremities, but consciousness was not disturbed, nor was there any anæsthesia or paralysis. Nausea, constipation, convulsions, and irregular breathing were also present.

Post mortem, a large growth was found in the cerebellum, whilst the whole spinal cord was enveloped, particularly on its posterior aspect, by a tumour-like mass of the consistency of brain-matter, which appeared to blend laterally with the arachnoid.

Case 6 (Schultz)[3].—The patient was a girl of 16, with a strong neuropathic history. Her chief symptoms were pain in the back, tingling in the hands, and gradually increasing weakness of the arms; later, tingling and weakness came on in the legs also. Headache then came on, but there is no note as to the optic discs. There was rigidity of the spine, and before death the right knee-jerk was lost, whilst the left was weak.

Post mortem, the cranial contents and their membranes were healthy. The sub-arachnoid space from the cervical region to the cauda equina was occupied by a mass of new growth covered by normal arachnoid. The new growth was nodulated and contained hæmorrhages; it ensheathed the cord (excepting in part of the dorsal region), but was more abundant on the posterior aspect than on the anterior.

Microscopically, the growth was an alveolar sarcoma, probably originating in the pia mater.

Case 7. (Hughes Bennett).[4]—The patient was a man, 48 years of age, whose illness began with shooting pains in the legs. The legs then became ataxic and weak, and soon similar defects were noticed in the arms. Both knee-jerks were lost; sensation in the feet was retarded and dulled, but elsewhere was good. There were no ocular signs. Death from uncontrollable vomiting.

Post mortem, there was some flattening of the convolutions. The choroid plexuses of the lateral ventricles were œdematous,

Sections were then taken through the enlarged posterior root ganglia of the cauda equina. All of those examined showed well-marked aggregations of cells identical in form and size with those constituting the growth in the brain. These cells were arranged irregularly in groups, occupying the open lymphatic spaces immediately around the ganglia and the roots of the nerves, particularly the posterior, as they neared the ganglion from the cord. There was no sign that any of the nerve-fibres or cells were destroyed in any of the specimens examined. It was clear that there was an early infiltration of the root-ganglia by tumour cells, and there could be little doubt that their focus of origin had been the mass of growth in the cerebrum from which the metastases had been wafted by means of the cerebrospinal fluid.

Case 3 (Coupland and Pasteur).[1]—The patient was a single woman, 22 years of age. Her only previous illness had been rheumatic fever.

Her fatal illness began with bilious headaches and morning vomiting; pain in the shoulders and arms, and in the occipital region of the neck. Some delirium and motor weakness were soon added to her other symptoms, and it was then found that well-marked optic neuritis was present, and that both external recti were paralysed; the knee-jerks were lost. There were no convulsions.

Post mortem.—There was an excess of cerebrospinal fluid. The medulla and pons were invested with a jelly-like growth in which both sixth nerves were imbedded. The theca was adherent to the cord at one or two points. The cord was ensheathed in its whole length by a semi-transparent soft growth, which was most abundant on the posterior and lateral aspects. Many nodules were present on the intraspinal nerve-roots. Microscopically, the tumour was a small round-celled sarcoma.

Scattered growths were also found on the arachnoid at the base of the brain.

Case 4 (Coupland and Pasteur).[1]—After a fall down stairs, a girl, 4½ years old, became irritable, developed a squint, lost her power of walking, had pain in the head, and later optic neuritis. There was no anæsthesia or local paralysis. Difficulty in raising the eyelids, weakness of the trunk muscles and dilatation of the frontal veins followed. Before death, both arms were totally paralysed, but the feet never completely lost their power. There were no convulsions.

Post mortem.—There was increased intracranial pressure and excess of fluid in the ventricles. Beneath the arachnoid covering

the inferior surface of the cerebellum was a layer of greenish material which penetrated into the nervous tissue for about half an inch. The middle lobe of the cerebellum was almost entirely replaced by soft vascular growth. Few scattered islets of tumour formed thin plaques at the base of the brain in the pia mater. Over the whole cord the pia was thickened posteriorly, the maximum thickness being about 6 millimetres. In places there was growth on the lateral and posterior aspects of the cord.

Microscopically, the growth was a soft sarcoma.

Case 5 (Ollivier)[2].—A boy, 11 years of age, had complained of attacks of headache accompanied by retraction of the head and recurring every six months. There was vomiting, pain in all four extremities, but consciousness was not disturbed, nor was there any anæsthesia or paralysis. Nausea, constipation, convulsions, and irregular breathing were also present.

Post mortem, a large growth was found in the cerebellum, whilst the whole spinal cord was enveloped, particularly on its posterior aspect, by a tumour-like mass of the consistency of brain-matter, which appeared to blend laterally with the arachnoid.

Case 6 (Schultz)[3].—The patient was a girl of 16, with a strong neuropathic history. Her chief symptoms were pain in the back, tingling in the hands, and gradually increasing weakness of the arms; later, tingling and weakness came on in the legs also. Headache then came on, but there is no note as to the optic discs. There was rigidity of the spine, and before death the right knee-jerk was lost, whilst the left was weak.

Post mortem, the cranial contents and their membranes were healthy. The sub-arachnoid space from the cervical region to the cauda equina was occupied by a mass of new growth covered by normal arachnoid. The new growth was nodulated and contained hæmorrhages; it ensheathed the cord (excepting in part of the dorsal region), but was more abundant on the posterior aspect than on the anterior.

Microscopically, the growth was an alveolar sarcoma, probably originating in the pia mater.

Case 7. (Hughes Bennett).[4]—The patient was a man, 48 years of age, whose illness began with shooting pains in the legs. The legs then became ataxic and weak, and soon similar defects were noticed in the arms. Both knee-jerks were lost; sensation in the feet was retarded and dulled, but elsewhere was good. There were no ocular signs. Death from uncontrollable vomiting.

Post mortem, there was some flattening of the convolutions. The choroid plexuses of the lateral ventricles were œdematous,

each lateral ventricle containing 4 cc. of clear yellow fluid. "In the substance of each frontal lobe, involving the anterior cornua of the lateral ventricle, was a circumscribed patch of softening, on the left side about the size of a hen's egg, and on the right side somewhat smaller. These extended inwards to the third ventricle and longitudinal fissure, downwards to the base of the brain and upwards to within one inch of the cortical substance." Microscopically, this area gave the appearances of cerebral softening.

The spinal dura was free, but the pia was thickened especially on its posterior aspect, most markedly below and gradually diminishing from below upwards. Posteriorly, the pia was studded with numerous tumours, more numerous below where they became confluent. The growths were attached to, and surrounded the posterior nerve roots. One sarcomatous mass was present in the floor of the fourth ventricle, and others were found on the under surface of the cerebellum.

Microscopically, the growth was a round-celled sarcoma.

Case 8. (Heubner).[5]—The patient was a girl with a good previous history. A short time after a fall on the arm, she began to experience cramps in the upper extremities, stiffness in the neck, headache, vomiting, disturbance of consciousness, and rapidly progressive loss of eyesight; hydrocephalus came on and steadily increased; there was pain in the upper arms, and flaccid palsy of the lower extremities with loss of knee-jerks at a late stage.

At the autopsy, there was a marked hydrocepalus. The membranes of the brain and spinal cord were greatly thickened, and six separate tumour masses, afterwards found to be gliomata, were present in the dorsal region of the cord. Secondary degeneration in the posterior columns was present.

The author concludes that the gliomata of the cord developed first, and that there was a secondary meningitis spreading up to the brain with a resulting hydrocephalus. He admits the difficulty of supposing a primary cord lesion, with symptoms which at the onset were purely cerebral.

Case 9. (Pels Leusden).[6]—In December, 1893, a man, aged 22 years, began to suffer from "rheumatic" pains in the back and legs; these gradually increased, and then the left leg became stiff and weak. Paralysis of the right leg followed, and then he began to complain of headache, intercostal neuralgia, and failing vision. By May, 1895, he had also difficulty in micturition and defæcation, and on admission to hospital he was very cachetic. There was now found to be complete paralysis and anæsthesia of both legs; a slight convergent strabismus was present. In the right

eye, there were opaque nerve fibres at the edge of the optic disc, and it was impossible to be certain whether optic neuritis was present, but it was undoubtedly present in the left eye.

Post mortem, a tumour was found infiltrating the pia-arachnoid at the base of the brain and around the spinal cord, occupying practically the same positions as that figured in Case 1. Microscopically, the growth was a glia-sarcoma.

Case 10. (Fischer)[7].—An eight-year old girl began to have a girdle pain in the abdomen, and in the small of the back, and weakness of the legs. When examined in June, 1899, there was complete flaccid paraplegia, paralysis of the bladder, and complete loss of all forms of sensation in the legs and in the trunk below the fifth dorsal root on the right, and the seventh on the left. There was no pain on percussion over the vertebræ.

In November there was commencing weakness of the upper extremities, and these were completely paralysed by February, 1900. The right pupil was now larger than the left, and there was congestion of both optic discs with tortuosity of the veins. Death occurred in March, 1900.

At the autopsy, the fifth to the tenth vertebræ were attached to the spinal cord which was greatly thickened. In the lumbar region a growth had made its way forwards through the bodies of the vertebræ to form a lump in the abdominal cavity the size of a hen's egg. The whole of this region of the cord was replaced by growth. The medulla, the base of the fourth ventricle, and the corpora quadrigemina were all infiltrated with growth. In the base of the third ventricle between the optic tracts was a translucent greyish neoplasm, whilst a second similar one was present in the left hemisphere, involving the head of the caudate nucleus. The growth was a glia-sarcoma, and the author considered that the growth in the cord was the primary one.

Case 11. (Pfeiffer)[8].—The patient was a 64 year old woman, with no inherited tendencies. For six months had suffered from cough with muco-purulent expectoration, wasting, no hæmoptysis, no night-sweats. At the same time, pains were present in the outer side of the right thigh spreading down to the foot.

On first admission (March, 1889), pulmonary tuberculosis was found at both apices, and there was present sciatica on the right side. Otherwise normal.

On second admission, three months later, besides the pulmonary condition there was present right-sided optic atrophy, and the right knee-jerk was absent; soon afterwards the left knee-jerk was also lost. A clinical diagnosis of pulmonary tuberculosis and incipient tabes was made.

Post mortem, there was found in the nervous system a "tuberkel im linken Corpus caudatum." There was great thickening of the arachnoid, of the extra-medullary nerve-roots of the spinal cord, and infiltration of the sheaths of the optic nerves, especially of the right, with epithelial cells. There was hyaline degeneration of many of the blood-vessels, and, by the Weigert method, degeneration in the posterior columns of the cord. There were also irregular deposition in and around the medulla. There is no report of the microscopic characters of the tumour in the caudate nucleus, nor was the cerebrum examined. In discussing the case, the author admits the strong resemblance of the epithelial formation to new growth, but on account of its widespread situation, concludes that it is a congenital anomaly. He remarks that the cylindrical epithelium reminds one forcibly of that of the central canal, and thus supports the idea of a congenital abnormality.

In many of the cases here recorded it will be noticed that a tumour mass was present which more or less ensheathed the whole spinal cord, whilst in some of the cases the meninges at the base of the brain were also infiltrated with sarcoma. In recording their work, most of the authors have attempted to fix upon some point as the origin of the growth. Coupland and Pasteur, in their summary conclude that the seat of origin in all the cases they considered (viz., Cases 3, 4, 5, 6 and 7) was spinal, except in the case reported by Ollivier (No. 5), which they consider to have been primarily cerebellar. They assume that the growth spread by continuity from the spinal pia both upwards and downwards, thus eventually involving the pia at the base of the brain late in the disease. Heubner (Case 8) also insists upon the primary spinal origin of the tumours in his case, although all the clinical symptoms seemed to show that the disease in the brain arose first. Fischer (Case 9) also considered that in his case the disease was primarily spinal, and that the cerebral membranes were secondarily infected, but he does not give any adequate explanation of how this occurred. It seems to me that it is far more likely that in most, if not in all these cases, the primary growth was situated high up in the nervous system. In my cases it is quite clear that the primary growth was in the frontal lobe of the brain, and that a rupture of the tumour had

caused infective particles to become disseminated throughout the central nervous system by means of the cerebrospinal fluid. In this way it is easy to trace the path of the infective particles flowing with the stream instead of against it, as would necessarily be the case if the growth originated in the spinal pia. If we assume that the cerebrospinal fluid secreted by the choroid plexuses of the lateral ventricle became charged with tumour particles, each capable of continued growth in a suitable nidus, it is then easy to understand how the whole track of the fluid down the ventricular system of the brain and in the subarachnoid spaces of the brain and cord would become the seat of a mass of secondary sarcoma. On this assumption, the solid particles would be filtered off from the fluid as it left the cerebrospinal canal, particularly along the course of the posterior roots near the ganglia, whilst other particles would be left more or less regularly in the lining of the ventricles of the brain and in the subarachnoid space around the cord. In each region the subsequent development of the growth would give rise to the appearances which have been described.

A strong argument in favour of the intracranial origin of these masses of growth is furnished by the analogous condition of tubercular meningitis. In a case of tubercular "tumour" which recently came under observation, in which death occurred from tubercular meningitis, it was noted that the involvement of the membranes at the base of the brain and of the pia mater of the cord corresponded exactly to that of the growth in Case 1. Here the primary focus was a "tumour" (tubercular), about the size of a hazel-nut, on the exposed under surface of the cerebellum, and, though it had not ruptured, it was clearly the point of origin of the meningitis. There could in this case be no question of spread by continuity from below; the infection was clearly one which spread by the cerebrospinal fluid.

It may be argued that in the first case which I record (Case 1) the rupture of the tumour may have given rise to the secondary growths, but that without rupture no such dissemination would have occurred. Again I fall back on an analogy. In those cases of disseminated carcinoma of the

peritoneal cavity, where the secondary growths are small and scattered (not continuous) over the parietal and, to a less extent, over the visceral peritoneum, there can be no doubt that the original focus has infected the peritoneal fluid, and that the dissemination has occurred by the streaming of these particles of malignant growth towards the stomata, where they are arrested and form new growths. And yet in these cases it is the exception rather than the rule to find a carcinomatous gland, or other tumour, which has actually ruptured. It may be that the rupture has been a small one, and that it has healed before the death of the patient.

The main argument against this mode of origin and distribution has been that a large growth has not always been found in the brain. Such a growth was present in my first case in such a marked degree that the most sceptical could hardly doubt that it was primary. In Case 4 there is a suggestion that the primary growth was in the vermis of the cerebellum roofing in the fourth ventricle; in Case 5, again, the primary growth was in the cerebellum; in Case 7, which differs from the others in that the growths did not become universally distributed over the spinal cord, but in no other essential particular, it seems to me more than likely that the softening in the frontal lobe was in reality a softened primary growth, and that this softening was the direct cause of the dissemination. In Case 8 I find it difficult to believe that we are dealing with a true meningitis spreading up to the brain as a result of the tumour.

The difficulty of distinguishing chronic inflammatory tissue from sarcoma is such that the microscope alone is not always sufficient, and whatever the histological appearance one would be inclined to think that the "meningitis" in this case was in reality a diffuse sarcomatous infiltration of the membranes.

In most of the recorded cases, the growth around the cord, though often coarsely uneven in thickness, does not suggest that at one point it has been present for a long time before growth occurred elsewhere; with one exception (Case 10) the spinal cord is not destroyed at any point, nor is there any great single mass of growth in the spinal region.

It, therefore, seems far more rational to look further afield for the point of origin of the tumour, and to see if the spinal mass is not all of the same age, and secondary to some other growth.

All of the cases are of a sarcomatous nature, but they vary considerably in malignancy. It is one of the most remarkable features that despite their almost complete involvement of the spinal cord, they rarely invade its substance, and then only to a very slight extent. The sarcoma seems to have a special affinity for the pia mater, growing easily amongst its tissue spaces, but not easily bursting through the membrane to invade the cord. In one of the cases, this invasion had occurred in several segments of the cord, and Fisher, therefore, concluded (Case 10) that this was the point of primary growth; that this growth was one of unusual malignancy is perfectly clear, but I am much inclined to doubt whether the growth was primary in the membranes of the spinal cord. It is difficult on that assumption to see how a growth could have occurred, as is described, in the head of the caudate nucleus, and how the medulla and mid-brain could be involved in growth on their ventricular surfaces. It could not be by dissemination through the cerebrospinal fluid, for the flow is in the downward direction, and such a spread would have to be upwards; so that the only way remaining would be by continuity—and the continuity is obviously frequently broken.

One objection may be raised to the suggestion that these growths have an intracranial origin, viz., the absence of intracranial symptoms until late in the disease. It must be remembered, however, that the primary growth usually seems to originate in the ependyma lining of the ventricles, the third and lateral particularly, and that if, for instance, the caudate nucleus were the point of involvement, we should expect no symptoms until the growth had attained a considerable size, and had begun to give rise to the general signs of intracranial tumour. But in such a position, with the cerebrospinal fluid washing directly over the growth, it is highly probable that dissemination would occur before the primary growth had given rise to any symptom of its

existence, and it might easily happen that the first signs should be shown by the secondary growths.

The relative non-malignancy of these growths is also shown by the way in which the intraspinal nerve-roots are affected; as a rule, there is much infiltration of the various bundles of fibres, and even separation of individual fibres, without marked destruction of the latter. It becomes an interesting speculation as to whether there do not exist types which are still malignant in the sense that they give rise to metastases, but which are less rapid in their growth and exhibit more innocent histological characters. It is possible that some of the cases of central (intracranial and intravertebral) neuro-fibromatosis are of this nature. The situation of the nodules of growth in these cases is similar—chiefly on the nerve-roots (especially of the cauda-equina), and less frequently in the pia of the spinal cord. It is true that we usually denominate them as "innocent" growths, and yet, single growths arising in the posterior root sheaths are known to recur locally if in removal a fragment of the root-sheath is left behind. The danger of recurrence under a malignant form after operative interference has so often been remarked upon that one cannot help thinking that the original growth has at least potential malignant characters, and that it may happen that in the course of its growth it may at times disseminate particles which can grow in a suitable habitat. For such growths, it seems clear that there is a special predilection for the sheaths of nerves, and that even were the infective particles to be wafted to the lungs or liver, they might be unable to grow there on account of unsuitable surrounding conditions. It is thus possible that certain of the cases of central neuro-fibromatosis may be regarded as being the result of dissemination of secondary infective particles from a single neoplasm higher up in the central nervous system, rather than as the expression of a series of congenital malformations.

Another point of interest in these cases has been the diffusion of the growths in the ventricles of the brain. In Cases 1, 2, 10 and 11 there were present discrete masses of growth in the lining of the third, lateral and fourth ventricles;

in none of the cases was the growth very obvious, and it is possible that similar growths have been present, but have not been recognised in some of the other cases. The occurrence of such growths in Cases 1, 10 and 11, in association with involvement of the arachnoid and pia around the spinal cord can hardly be regarded as a mere coincidence, and it seems only rational to suppose that both have occurred as metastases from some primary growth. Thus, in Case 1 the tumour in the frontal region could give rise to particles which might infect the ventricles, whilst other particles might easily pass with the cerebrospinal fluid through the foramina in the roof of the fourth ventricle, and so reach the cisterna magna, the subarachnoid spaces of the spinal cord, and, finally, the posterior root ganglia, where the last of such infective particles would be filtered off. In Case 10 it is easy to understand the course of the disease if we assume that the growth at the base of the third ventricle, or that in the caudate nucleus, was the primary one, and that one of the early metastatic growths grew sufficiently rapidly to give rise to a large tumour at the time of death ; whilst a growth in the caudate nucleus in Case 11 would adequately explain the method of origin of the various growths found at the autopsy.

From a clinical point of view, the symptoms described have naturally been very varied, partly on account of the disseminated character of the growth, and partly because in some cases there has been an obvious growth, or in some cases several growths, within the brain. In any case there is almost constantly present in the later stages some hydrocephalus, clearly secondary to the growths. This may be caused either by blocking of the outlet from the skull of the cerebrospinal fluid or by blocking of a large number of the possible outlets from the spinal theca. It will therefore be seen that the headache, vomiting, and optic neuritis, if they occur at all, may either arise at an early stage before the symptoms due to large local growths have shown themselves, or may only be present as the terminal phase of the condition

Considerable discussion was devoted by Coupland and

Pasteur to the question as to whether convulsions should be expected, and they thought that the absence was one of the most characteristic phenomena of the condition. But, from a consideration of the other cases, it is clear that they do occur, although not in the early stages. This is probably due to the fact that the cerebral cortex is not as a rule directly involved, the growth tending to affect the ventricular lining and the pia mater rather than the cerebral substance. The convulsions which do occur are probably in most cases due to the rise in intracranial pressure which comes on late in the disease, although in Case 1 they may have originated in the diseased frontal cortex.

It will be noted that peripheral pain along the area of supply of the involved nerve roots was present in most of the cases, but rarely reached a severe grade such as is usually seen in single root tumours. This may be due to the fact that the nerve roots are not severely compressed by the rather soft encircling growth as they are by the less malignant and often fibrous single tumours. It is, however, more likely that the youth of the patients is a better explanation, for the only middle-aged patients in the group suffered so severely from shooting pains that they presented the clinical picture of tabes dorsalis.

In general it remains impossible to give any accurate diagnostic signs of the condition; but it seems clear that if a patient who is known to present the signs of unlocalisable cerebral tumour develops well-marked pains in the limbs and trunk, with early loss of deep reflexes and rapid wasting, the presence of disseminated growth in the spinal pia and posterior root ganglia should be suspected. In such cases it would be well to examine also the ventricular lining in case the less obvious gelatinous growths which occur in this situation may be overlooked.

Conclusions.

(1) If a growth originates in the caudate nucleus or in any other part of the brain and eventually finds its way to the lining of the ventricular cavities, it may "infect" the cerebrospinal fluid. Metastatic growths may then occur in the

following situations: (1) In the lining walls of the lateral, third or fourth ventricles; (2) In the meninges (subarachnoid) at the base of the brain; (3) In the pia mater and arachnoid around the spinal cord; and (4) in the posterior root ganglia, particularly of the cauda equina.

(2) In all probability diffuse sarcomatous infiltration of the spinal pia-arachnoid, which is occasionally found post-mortem, is in all cases the expression of a sarcomatous infection of the cerebrospinal fluid as a result of some primary growth which lies exposed to the stream of the cerebrospinal fluid high up in the nervous system.

(3) The regions occupied by such secondary growths are exactly the same as those inflamed by tubercular meningitis after the rupture of a tubercular "tumour" of the brain.

Bibliography.

(1) Coupland and Pasteur. *Trans. of Path. Soc.*, xxxviii., p. 26.
(2) Ollivier. "Traité des Maladies de la Moelle Epiniere" (1837, T. 2, p. 490).
(3) Schultz. *Arch. f. Psych.*, 1885, p. 542.
(4) Hughes Bennett. *Clin. Soc. Trans.*, vol. xviii., p. 168.
(5) Heubner. "Ueber ein Fall von Multiplen Rückenmarksgliomen, Meningitis und Hydrocephalus." *Verhand. der Gesell. für Kinderheilkunde zu Aachen*, 1900.
(6) Pels Leusden. *Ziegler's Beiträge*, Jahrg. xxiii., S. 96.
(7) Fischer. "Ueber ein selten mächtig entwickeltes Glia-sarcomatodes des Rückenmarks." *Zeitschrift f. Heilkunde*, xxii.
(8) Pfeiffer. "Ueber eigenartige Veränderungen in der Arachnoides, der extramedullären Rückenmarkswurzeln und der beiden Nervi optici." *Deutsche Zeit. für Nervenheilkunde*, 1894, B. 5, p. 45.

SOME HITHERTO UNDESCRIBED SYMPTOMS IN ANGINA PECTORIS.

BY G. A. GIBSON, M.D., D.SC.EDIN., LL.D.ST.AND., F.R.C.P.EDIN.

A Clinical Lecture delivered in the Royal Infirmary.

ALTHOUGH angina pectoris was the subject of one of my clinical lectures last term, the subject is so important as to merit further study. We have, moreover, at present in our wards the most interesting example of the affection which it has ever been my good fortune to observe. The complex of symptoms presented by this patient is so extraordinary that it seems my duty to lay the facts before you in full detail. They will furnish an occasion for amplifying the remarks previously made on the modern conception of the condition.

A labouring man, aged 45, born in Leitrim, but resident for some years in Edinburgh, was admitted to my ward on account of pain in the chest and left arm. His father lived to the age of 85, and was said to be subject to rheumatic pains. His mother died, when 33 years old, of puerperal causes. The patient says he was never addicted to alcoholic excess, seeing that for him it was too expensive a luxury. He has been in the habit of smoking about two ounces of strong tobacco every week. His home conditions are healthy and comfortable, but his work is rather severe. When 21 years old he suffered from some fever, as to the nature of which he is ignorant. In more recent years he had an attack of pneumonia. The commencement of the present illness appears to have been about six weeks ago, and it seems to have begun with breathlessness on exertion; the actual onset of the pain in the chest and arm was five weeks before admission. Its first appearance was when he was in a train on the way to his work. He was able to perform his

duties for three days, but since then has been unfit for any exertion. The pain has persisted continuously ever since its first onset, but two weeks after it began it underwent a change. At first it was confined to the region of the left scapula, but it afterwards began to shoot down the left arm, and at the same time it showed paroxysmal exacerbations.

The present condition of the patient is as follows:—He is a strongly-built man, 5 ft. 7 in. in height, weighing 10 st. 1 lb., with a healthy, florid complexion.

The circulatory system gives evidence of considerable alteration. The patient, as a rule, has a high complexion, except during a paroxysm of pain, when he becomes paler. There is some dilatation of the venules on the cheeks and nose, and the temporal arteries stand out in a sinuous fashion, although there is no excessive pulsation in them or in the carotids. The superficial arteries are thick and tortuous. The arterial pressure is high at all times; measured by Hill and Barnard's sphygmometer it varies between 160 and 170 mm. Hg. During the paroxysms of pain the pressure

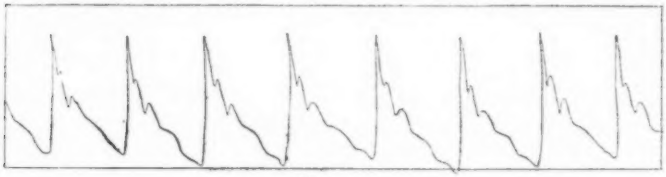


FIG. 1.
Usual pulse. Pressure 4 oz.

rises considerably, but for obvious reasons it is then difficult to obtain an exact measurement. The pulsation is perfectly regular and not unduly frequent; each individual pulsation

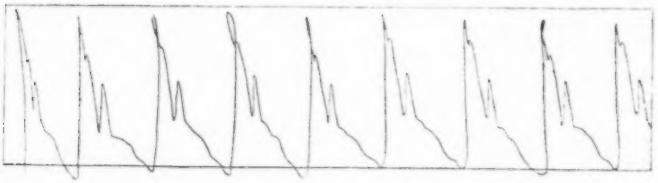


FIG. 2.
Usual pulse. Pressure 5 oz.

is of large amplitude and is well sustained. Tracings of the ordinary pulse are given in figs. 1 and 2.

The Junior House Physician was in the act of taking some sphygmographic tracings when one of the paroxysms of pain set in, and he found that with the increase of pressure there was a characteristic reduction in the size of the pulse, with some acceleration, as shown in fig. 3, while,

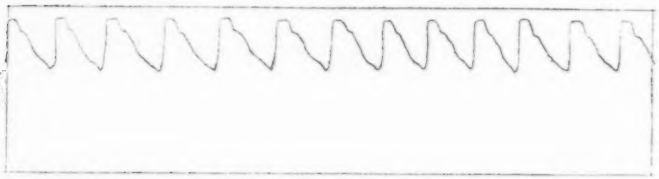


FIG. 3.
Pulse during paroxysm of pain. Pressure 3 oz.

after the use of nitrite of amyl and the relief of pain, the pulse became large, bounding, and still more frequent, as

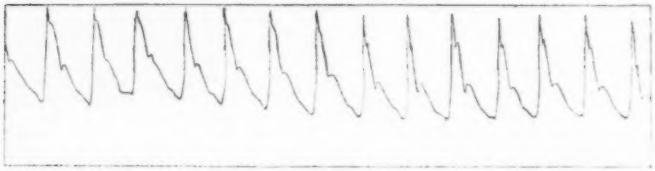


FIG. 4.
Pulse after nitrite of amyl. Pressure 3 oz.

in fig. 4. The case, in respect of the pulse, therefore, conforms to the type of angina with spasm of the arterioles.

On inspection, the apex beat is seen in the fifth intercostal space, about the mammillary line. There is no other impulse visible in any part of the præcordia or epigastrium. On palpation, the apex beat is found to be situated $4\frac{1}{2}$ in. from mid sternum. The impulse is forcible and without any thrill. The superior border of the heart on percussion is found at the upper edge of the third left costal cartilage.

The left border is $4\frac{3}{4}$ in. and the right is 2 in. from the middle line. The heart sounds are perfectly clear, but the aortic second sound is much accentuated.

The most careful investigation of the chest fails to give any evidence of other disease, and the X-rays, employed both by screen and by skiagraphy, show a perfectly normal state of matters at the base of the heart.

The patient complains of an extensive and constant pain, most severe, as a rule, over the back, where it extends down as far as the ninth intercostal space. It reaches up to the back of the neck, and passes round its side so as to reach the left clavicle, which it crosses, and extends down the middle

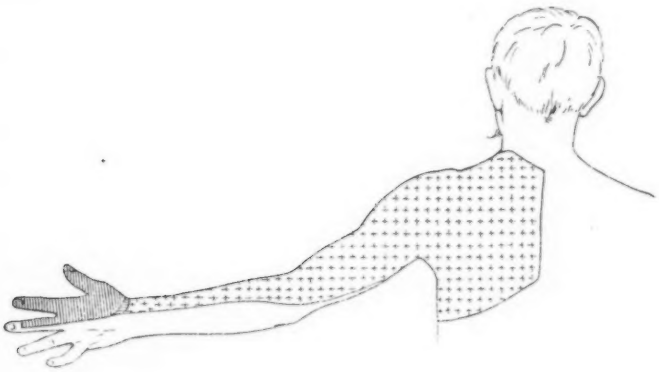


FIG. 5.

Distribution of sensory changes posteriorly. The crosses denote hyperæsthesia; the horizontal shading analgesia without anæsthesia; the vertical shading analgesia with anæsthesia.

line to the eighth intercostal space, from which point it becomes continuous with the pain at the back by passing round the inferior axillary region. From the shoulder it extends down the outer aspect of the arm and forearm so as to reach the thumb, index and middle fingers. The exact boundaries of the pain can be seen in the figs. 5 and 6, drawn from photographs taken after the areas had been marked on the skin. When the paroxysms of pain occur, the boundaries appear to the patient to be somewhat enlarged, and it

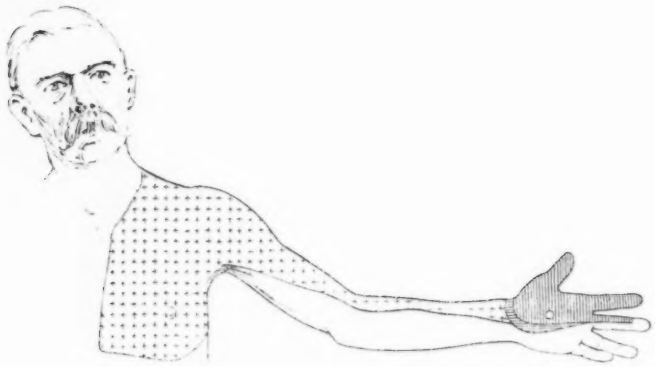


FIG. 6.

Distribution of sensory changes anteriorly. Explanation as in fig. 5.

shoots up the left side of the head so as to involve the cranial vault as far as the middle line, and includes most of the arm and forearm.

On testing the sensibility of the surface, a most interesting group of phenomena can be discovered. Over the whole

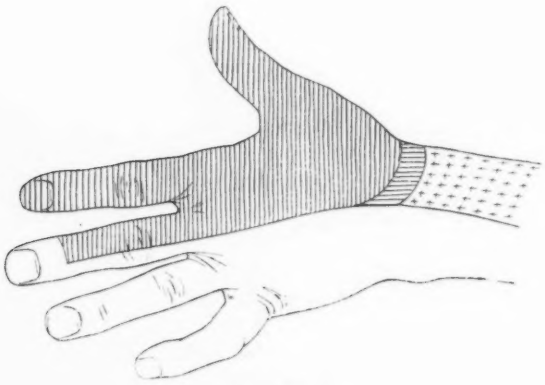


FIG. 7.

Sensory changes in back of hand. Explanation as on fig. 5.

of the area marked with crosses there is a high degree of hyperæsthesia to every form of stimulus, but over the radial portion of the hand there is absolute anæsthesia, bounded along part of its edge by a small zone of analgesia. The anæsthesia is complete to every kind of stimulus, even to the most intense we can employ; the analgesic area is quite sensitive to all ordinary tests, but it is impossible to elicit the least sensation of pain. The exact distribution of this is marked upon the patient by horizontal and vertical

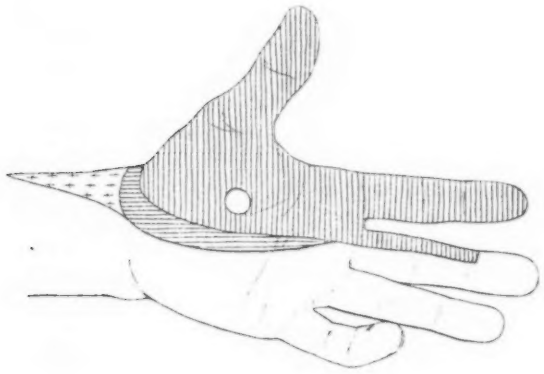


FIG. 8.

Sensory changes in front of hand. Explanation as on fig. 5.

lines, and it may be seen in figs. 7 and 8. The anæsthesia is profound to every form of stimulus, but there is one small point about the size of a three-penny piece at the junction of the thenar eminence and the palm of the hand in which the patient feels perfectly well. Probably, as Professor Cunningham has suggested to me, this may be due to a communicating branch from the ulnar nerve.

Another point of interest is that the sterno-mastoid, trapezius, pectoralis, deltoid, biceps and supinator muscles of the left arm are much more tender than those of the right. There is no other sensory disturbance and the special senses are intact.

Now, before leaving the afferent nervous system, it is necessary to point out two divergences from the usual state of matters exhibited by our patient. One of these may be due to an anatomical peculiarity. It is the line of division separating the insensitive from the sensitive part of the palm. Instead of being bounded by the middle of the ring finger, as is usual in front, the line of demarcation of ulnar and median nerves runs along the middle of the middle finger, unless we may assume that a portion of the median nerve has escaped. The other departure from what is customary is much more difficult to understand. As will be observed from the outlines painted on the patient, the inner or ulnar aspect of the arm, forearm, and hand, is not affected, *i.e.*, a considerable area belonging to the segmental distribution of the seventh and eighth cervical and first dorsal portions of the cord is intact, while the areas above and below are deeply involved. Now the segments unaffected in our case are those usually most deeply involved. In view of the cilio-spinal effects about to be described, this fact is difficult to understand, and it seems to me to indicate a capriciousness of impression on the part of the spinal segments.

The organic reflexes are normal. The cutaneous reflexes are all present and appear to be within usual limits. The supinator and triceps jerks are increased in the left arm as compared with the right, and the myotatic irritability is increased in the deltoid and the other muscles on the outer aspect of the left arm and forearm. There is great weakness of the left hand, especially in the thumb and first and second fingers, so that the patient's grasp is practically restricted to the ring and little fingers. With the dynamometer the grasp of the right hand is 135 and of the left 50. There is wasting and loss of tone in all the muscles of the shoulder-girdle, arm, forearm, and hand on the left side. The right arm is in circumference 12 in., the left 10½ in. The right forearm is 10½ in., and the left 10 in. On testing the electric reactions an increased excitability is found in the muscles which are affected. The exact state of matters will be easily understood from the following table, in which the

measurements of faradism are expressed in the number of centimetres on the scale of the induction apparatus, while those of galvanism are given in milliampères:—

Faradism.

	R.		L.
Deltoid	2.7	...	2
Biceps	1·6	...	1·4
Supinator longus	1·9	...	1·2
Flexor sublimis	3·2	...	1·9

Galvanism.

	R.		L.
Deltoid	9 ccc. / 10 acc.	...	6 ccc. / 7 acc.
Biceps	5 ccc. / 6 acc.	...	3 ccc. / 4 acc.
Supinator longus ...	6 ccc. / 7 acc.	...	5 ccc. / 6 acc.
Flexor sublimis... ...	11 ccc. / 12 acc.	...	9 ccc / 10 acc.

One other point deserves consideration. The left eye is more prominent than the right, and the left pupil is larger than its fellow. This condition has been present throughout his residence in the ward, but it is more marked at those times when the pain gives him serious trouble. There cannot be a doubt, therefore, that there is irritation of the sympathetic nerve on the left side, involving the cilio-spinal fibres. It is interesting to observe that these phenomena are not accompanied by any alteration in the functions of the skin. There is no redness or increase of perspiration, but, as already mentioned, there is some general pallor of the whole countenance during an attack.

The diagnosis in this interesting case is early arterial sclerosis, probably implicating the coronary arteries, and accompanied by some interstitial changes in the myocardium —the combination, in short, which is so commonly attended by angina pectoris. But, as already remarked, there is a vaso-motor element in the attacks; this, as is universally recognised, is a symptom frequently met with.

Numbness of the arm, the forearm, or the hand, has often been observed in cases of angina pectoris, but complete anæsthesia, such as is present in this case, must be rare indeed; in fact, no instance even distantly approaching that just described, has ever come under my notice. Neither in the beautiful researches of Mackenzie,[1] nor of Head,[2] is there any reference to such conditions. In a private communication, however, the former states that he seemed to obtain anæsthetic areas when he first investigated the sensibility in angina pectoris, but that in late years he had failed to find them and concluded he had made a mistake.

The wasting of the muscles has been described both by Eichhorst [3] and myself,[4] and there is no difficulty in explaining such trophic results from powerful afferent impulses. They are strictly analogous to the conditions which are found in arthritic muscular atrophy. This has long been considered as due entirely to reflex influence, which has been fully discussed by Paget [5] and by Vulpian,[6] and the theory is strongly supported by the fact that in experimental joint lesions wasting of the muscles is prevented by previous division of the posterior spinal roots, which has been described by Raymond.[7] But there are some distinct differences between the wasting in this case and in that of arthritic muscular atrophy. In this patient, just as occurs in arthritic atrophy, there is considerable increase in myotatic irritability. But no case of arthritic dystrophy of muscles has ever been under my notice in which there was an increase in the electric irritability of the atrophied muscles. It is possible, however, that what we are dealing with is an early condition, and that a reduction in electric irritability may be a later phase.

In addition to providing a number of hitherto undescribed symptoms for our study, the patient seems to me of the deepest interest as giving proof of a means of communica-

[1] *Medical Chronicle*, 1892, p. 293; and *Lancet*, 1895, Vol. I., p. 16.
[2] BRAIN, 1893, vol. xvi., p. 1, and 1894, vol. xvii., p. 339.
[3] "Handbuch der Specieller Pathologie und Therapie," 1895, S. 231.
[4] "The Nervous Affections of the Heart," 1904, p. 18.
[5] The *Lancet*, 1873, Vol. II., p. 727.
[6] "Leçons sur l'Appareil Vaso-moteur," 1875, Tome 2, p. 327.
[7] *Revue de Médecine*, 1890, p. 374.

tion between the heart and the nerve centres which has hitherto received but scant attention.

While the vagus centripetal mechanism has been long recognised, that of the sympathetic system has been a matter of great doubt. In my Morison lectures,[1] the course of these tracts was fully described and the conclusions then reached may be now briefly summarised. Certain channels, proved by undoubted clinical facts to supply the course of afferent impulses, have not yet received support from any experimental evidence. The afferent impulses passing upwards in connection with the inhibiting and dilating mechanism are carried by the vagus nerve. The afferent impulses belonging to the accelerating and constricting mechanism must travel by the inferior cardiac nerve to the inferior cervical ganglion and thence by the gray rami to the seventh and eighth segments of the cord. It is possible that they may also pass round the annulus of Vieussens to reach the upper thoracic segments. But of afferent nerves belonging to the heart and passing to the spinal cord, there is no experimental proof. If we may judge afferent nerves by those of the visceral system of the abdomen, they are always medullated up to the posterior ganglion, and many of these medullated fibres are of large size. Reasoning by analogy it might be expected that the afferent cardiac fibres must also be medullated, but Gaskell,[2] when working at the fibres of the dog's heart was much struck to find that all the nerves from the annulus of Vieussens to the heart were non-medullated—no medullated fibres were traced to the depressor or the vagus nerve. He therefore concluded that the anatomical evidence confirmed the physiological, and that both pressor and depressor fibres reached the central nervous system by the vagus nerve. By means of a private communication he has, however, expressed his opinion to me that the spinal heart nerves are only a special example of the system of vaso-constrictor nerves, and therefore, if the blood-vessels possess

[1] *Op. cit.*, p. 32.

[2] *Transactions of the International Medical Congress, London*, 1881, vol. i., p. 254; *Philosophical Transactions, London*, 1883, vol. clxxiii., p. 993; *Journal of Physiology*, 1884, vol. v., pp. 46 and 362; *Ibid.*, 1886, vol. vii., p. 1.

afferent nerves, the heart, in virtue of its connection with that system must possess such nerves. He believes that there is evidence of the possession of spinal afferent nerves by the blood-vessels, while there is no evidence to prove that the blood-vessels possess any number of medullated fibres. His view is accordingly that it is possible, indeed probable, that afferent nerves, at least from the coronary vessels, should accompany the accelerator and so account for the clinical facts.

The real importance of this case, then, in my opinion, lies in the opportunity which it affords of giving a clear demonstration of certain facts regarding the afferent impulses from the heart; the symptoms exhibited by the patient allow the possibility of tracing out the course of these paths. In the diagram, fig. 9, the direction of these channels of communication is traced out. Passing up from the receptive end organ (*H*), by the cardiac nerves to the cervical ganglia (I *G*, II *G*, and III *G*), of the sympathetic, they pass in, as has been elsewhere shown by me,[1] by the gray rami communicantes (*PC*), to the posterior spinal roots, and thence run upwards in the ascending tracts of the spinal cord. In the cortex cerebri (*C*), the impulses which are produced give rise to sensation, but, as Head[2] puts it, "the sensory and localising power of the surface of the body is enormously in excess of that of the viscera, and thus by what might be called a psychical error of judgment, the diffusion area is accepted by consciousness and the pain is referred on to the surface of the body instead of on to the organ actually affected." The result is that the patient seems to feel uneasy or painful sensations over the præcordia, shoulder and upper extremity, as at *S*. That the cervical sympathetic is involved in this case cannot be denied on account of the ocular phenomena, and our patient may be taken as a proof of the contention that this system is the means of conducting afferent impulses from the heart. The figure (fig. 10) gives a diagrammatic representation of part of the cilio-spinal mechanism, showing the course of the spinal fibres

[1] *Op. cit.* p. 30.
[2] *Op. cit.*, vol. xvi., p. 126.

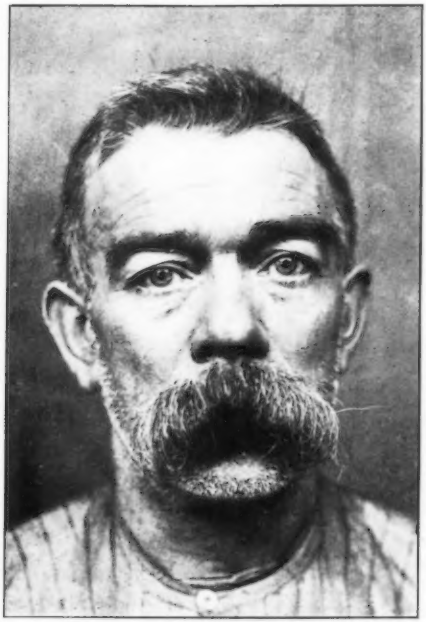


FIG. 9.

From a photograph of the patient, showing prominence of the left eye and dilatation of the left pupil.

from the pupil-dilating centre (*PD*) in the base of the brain down to the cilio-spinal centre (*CS*), and thence by the anterior spinal root and anterior or white ramus communicans (*AC*), to the sympathetic system, whence the fibres pass upwards to the radiating fibres of the iris (*DP*) to the muscle of Müller (*MM*), and to the non-striped

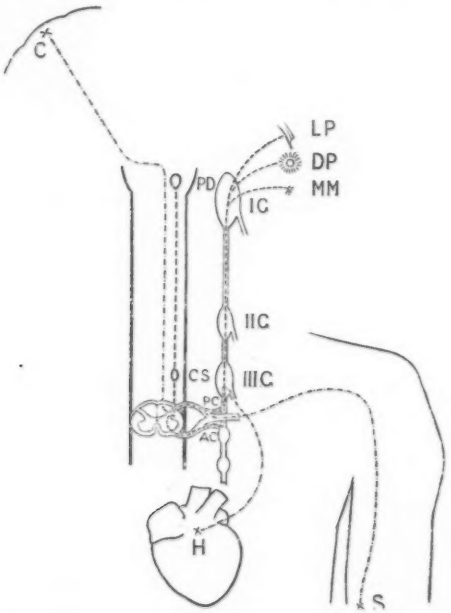


FIG. 10.

Course of impulses in the case. The dots and dashes indicate afferent—the dashes alone efferent tracts.

portion of the levator palpebræ (*LP*). In the construction of this diagram, a figure designed by Purves Stewart[1] has been very useful, and it is a pleasure to acknowledge its assistance. For untiring assistance in unravelling the details of this very interesting case my warm thanks are due to my House Physicians Dr. R. W. Johnstone and Dr. H. A. Stewart.

[1] *The Practitioner*, 1905, vol. lxxiv., p. 189.

Since the delivery of this lecture the patient has been almost continuously under observation, and the course of the affection must be mentioned. He was treated by iodide of potassium in ten grain doses three times a day, along with inhalations of amyl nitrite whenever a paroxysm of pain occurred. In the course of a week the severe attacks disappeared, and in less than a month the hyperæsthetic and anæsthetic phenomena were greatly diminished. But at the time of writing this additional note, four months after the patient came under treatment, there are still remains of the motor, electric and trophic changes, as well as some prominence of the left eye, and slight dilatation of the pupil. The hyperæsthesia is still slightly present, but in an almost inappreciable degree. Of the anæsthesia not a trace now remains.

ON A TRIGEMINAL-AURAL REFLEX IN THE RABBIT.

BY SIR VICTOR HORSLEY, F.R.S., F.R.C.S.,

Surgeon to University College Hospital, and the National Hospital for the Paralysed and Epileptic.

IT is well known that if a wild rabbit is feeding quietly its ears lie horizontally along its back, but that when startled it raises itself upright by extending its spine and then erects its ears.

This last movement of the ears has always been regarded as voluntary, *i.e.*, a high purposive act on the part of the animal to detect a source of danger. I have found, however, that such a protective elevation of the ear can also be produced as a simple reflex movement evoked by excitation of only one branch of the trigeminal nerve, namely, the infra-orbital. Of the following photographs, taken at the rate of $\frac{1}{100}$th of a second, the first shows in a young wild rabbit the ears in a position of rest. (I may remark parenthetically that such a little rabbit, even recently caught, will keep quiet for a long time in the warm hand if it is gently stroked, as shown in the photographs).

Photographs 2 and 3, a side and front view respectively, show that when the vibrissæ on the infra-orbital region are gently touched with the finger, or the skin of that part gently pressed (especially over the infra-orbital foramen), the homolateral ear alone is raised up vertically and somewhat rotated forwards.

Photograph 4 shows that the elevation of the ear is tonic and persists for several seconds after the stimulus is removed; the ear then slowly subsiding to the horizontal position.

If the animal was startled and resented the touch of the finger, the contralateral ear was also raised, and the rabbit

escaped as shown in Photograph 5, where the hind quarters are shown rising in the action of leaping.

It being obvious that the psychical stimulus of fear caused the bilateral general reaction, I tested other afferent paths when the rabbit was quieted. The results were wholly negative except as regards auditory stimuli. Thus, Photograph 6 shows the ears of the rabbit quite quiet although a ray of direct sunlight is reflected on to its eye; the only result being some protective contraction of the orbicularis oculi muscle. Similarly touch or pressure stimuli applied to the other parts of the body were without effect on the ear.

Pricking of the ears being undoubtedly associated with an appreciation of danger, it is reasonable that it should occur when the infra-orbital region, and especially the vibrissæ, are touched; since a rabbit in escaping from danger encounters numerous obstacles as it runs in the depths of the burrow, or through bushes and grass in the dark, and therefore must largely rely on contact impressions from its face.

Though most active in the wild animal I have obtained this reflex also in tame and albino rabbits, even whether partly lop-eared or no.

Next, as regards the localisation of this reflex movement, the phenomenon is analogous to the representation of the movements of the ear in the inferior colliculis (corpus quadrigeminum posterius), and that the motor path is the same for the auditory and infra-orbital reflexes, inasmuch as the movement executed by the pinna is identical, namely, elevation with rotation so as to present the conchal concavity forwards and outwards.

On the question of the localisation of movements of the ear, Ferrier[1] was the first to find that excitation of the anterior colliculi produced, in addition to movements of the eyes, marked "retraction of the ears," and that the same occurred on stimulation of the posterior colliculi.

V. Bechterew[2] similarly obtained by excitation of both the anterior and posterior colliculi turning of the contralateral ear outwards, in addition to conjugate deviation of

[1] "Functions of the Brain," 2nd Ed., 1886, p. 166.
[2] *Archiv. fur Physiologie*, 1884, vol. xxxiii., p. 413.

FIG. 1.

Animal at rest; ears horizontal.

FIG. 2.

Infra-orbital vibrissæ touched with finger-tip; homolateral pinna rising.

FIG. 3.

Front view of elevation of homolateral pinna only when infra-orbital region touched.

FIG. 4.

Tonic maintenance of the elevation of the pinna for a few seconds after the withdrawal of the stimulation.

FIG. 5.

Both ears pricked up as the animal is excited and escapes.

FIG. 6.

Ears horizontal, although ray of direct sunlight is flashed on to eye; example of absence of reaction of the pinna to certain other kinds of stimuli.

the eyes to the opposite side, &c. In a later paper[1] he stated (p. 711) that after unilateral destruction of the posterior colliculus in a rabbit the animal did not prick up the contralateral ear nor turn towards a source of sound.

Prus,[2] in his valuable paper on excitation of the colliculi in dogs, describes the movement of the ear in connection with stimulation of the anterior colliculus only.

Ziehen's [3] observations also coincide with the foregoing results, as also do certain observations by Thiele, which will shortly be published.

During recent years I have made many observations on excitation of the colliculi, anterior and posterior, especially in conjunction with Dr. Magnus during 1904; and in the cat, dog and monkey have observed, in confirmation of Ferrier, that faradic excitation of the outer surface of the posterior colliculus evokes, besides the rotation downwards of the homolateral eye (fourth nerve effect), marked rotation of the pinna of the contralateral ear. The same result was also obtained after section (whether made immediately or three weeks before the excitation) of the intercollicular commissure. The localisation of the phenomenon is therefore exact. Not infrequently the homolateral rather than the opposite ear moves, but the character of the movement is the same, viz., rotation for better audition, an action very different from the flattening and setting back of the pinna as a protection in fighting, which latter can be obtained from a wide area of the cortex cerebri in all animals, notably the ungulata. As, of course, the representation of the cochlear nerve in the posterior colliculus is now definitely established, the similar association with the fifth nerve of the "auditory" movement of the pinna, which evidently is of a defensive and useful character, is easy to understand.

[1] *Neurologisches Centralblatt*, 1895, vol. xiv., p. 706.
[2] *Wiener Klinische Wochenschrift*, 1899, No. 45, p. 1,124.
[3] *Archiv. fur Psychiatrie und Nervenheilkunde*, vol. xxi., p. 863.

A NOTE ON THE CONDITION OF THE TENDO ACHILLIS JERK IN DIPHTHERIA.

BY J. D. ROLLESTON, M.A., M.D.OXON.

Assistant Medical Officer at the Grove Fever Hospital of the Metropolitan Asylums Board.

THE condition of the tendo Achillis jerk in diphtheria, though alluded to by a few authors (1), has not received the same attention that has been given to the reflex in the case of sciatica and tabes. The present paper is based on 100 cases of diphtheria in which the tendo Achillis jerk was investigated until the time that the patient was allowed to sit up. This period varied in length according to the severity of the case, the condition of the heart, and the presence or absence of any paralysis, from twenty to sixty-three days from the onset of the disease. The reflex was sought for in the recumbent position. In forty-seven cases the Achillis jerks were affected to a varying degree; they were completely lost in twenty cases, and rendered sluggish in twenty-seven. In the remaining fifty-three cases they remained active throughout the period of observation, which in no case was less than six weeks, and in several cases was prolonged to three months or more.

From this it will be seen that though the Achillis jerks are affected in a large proportion of the cases, this affection is not so frequent as in the case of the knee-jerks. Owing to the greater liability of the diphtheritic toxin to impair the latter, affection of the Achillis jerks was never found without a corresponding or relatively greater impairment of the knee-jerks.

In Hector Mackenzie's (2) cases, the knee-jerks were lost in 80 per cent., *i.e.*, fifty-one out of sixty-four cases. In my own cases they were affected in sixty-nine, lost in thirty-one, sluggish in thirty-eight.

The higher percentage of Mackenzie's figures is probably due to the fact that his cases included a larger number of severe attacks than my own series, and that their course was not modified by serum. Early use of antitoxin, as I have shown elsewhere (3), diminishes the risk of subsequent paralysis. The same probably applies to affection of the reflexes.

As will be seen from Table I., the frequency and degree to which the Achillis jerks were affected bore a direct relation to the character of the initial attack, thus exactly corresponding with the frequency of the most characteristic complications of diphtheria, namely, paralysis and albuminuria (Table II.).

TABLE I.

Classes I. to VI. represent various degrees of severity of the faucial attack.

		Condition of Achillis jerks.		
	No. of Cases.	Lost.	Sluggish.	Not affected.
Class I.—Very severe	15	9	5*	1†
,, II.—Severe	22	7	5	10
,, III.—Moderately severe ...	9	2	5	2
,, IV.—Moderate	23	2	5	16
,, V.—Mild	28	0	6	22
,, VI.—Very mild	3	0	1	2

* One died on tenth day, and one on eleventh day. † Died on fourth day.

This table clearly shows that not only is affection of the Achillis jerks commoner in the severe than in the milder forms of the disease, but that in the first two classes in which Achillis jerks were affected at all, the majority were completely lost; the minority were rendered sluggish only. In classes III. and IV. exactly the reverse holds good. In the last two classes the Achillis jerks were not lost in a single instance.

TABLE II.

Showing incidence of paralysis and albuminuria in 100 cases where the Achillis jerks were investigated.

	Paralysis cases.	Severe forms only.	Albuminuric cases.	Persistent albuminuria, *i.e.*, for three weeks or more.
Class I.	... 13 = 86·6 %	... 11 = 73·3 %	... 14 = 93·3 %	.. 6 = 27·2 %
,, II.	... 9 = 40·9 %	... 3 = 13·6 %	... 11 = 50·0 %	... 0 = 0
,, III.	... 2 = 22·2 %	... 0 = 0	... 4 = 44·4 %	... 1 = 11·1 %
,, IV.	... 4 = 14·2 %	... 1 = 4·3 %	... 8 = 34·7 %	... 1 = 6·6 %
,, V.	... 1 = 3·5 %	... 0 = 0	... 4 = 14·2 %	... 0
,, VI.	... 0	... 0	... 0 = 0	

Further analysis of the cases will show the relation of the affection of the reflexes to paralysis. Thus, out of twenty cases in which the Achillis jerks were lost, in fourteen (70 per cent.) paralysis occurred, and in twelve was severe.

Among twenty-seven instances in which the Achillis jerks were sluggish, there were seventeen cases (62·9 per cent.) of paralysis, of which eight were severe. In those cases where the knee-jerks alone were affected, the incidence of paralysis was much less. Thus, in thirty-one where the knee-jerks were lost, there were seventeen cases of paralysis (54·8 per cent., of which thirteen were severe. Of thirty-eight cases in which the knee-jerks were sluggish without being lost, there were twelve paralysis cases (31·5 per cent.), of which eight were severe.

In all the cases where definite paraplegia was present knee-jerks and Achillis jerks were lost; but in some, where from the severity of the initial attack loss of motor power might have been expected, the patient was able to walk alone from the first, with but slight ataxia and Rombergism, although the knee and Achillis jerks were completely lost.

This abortive condition, to which Aubertin and Babonneix have given the name of "*paraplégie diphtérique fruste*," is characteristic of the great majority of diphtheritic palsies, as I have illustrated elsewhere (3).

In thirty-two cases of loss of motor power, varying in degree from complete paraplegia to an ataxic gait combined with Rombergism, the Achillis jerks were affected twenty-four times. In thirteen of these they were completely lost; in eleven they were sluggish. The date at which the Achillis jerks were affected varied in different patients.

In the great majority of my patients, viz., thirty-two out of forty-seven, the Achillis jerks were already affected to a certain degree on admission to hospital. In eight of the thirty-two they were completely lost; in twenty-four they were sluggish; 50 per cent. of the knee-jerks were already affected on admission, fifteen being lost, thirty-five being sluggish.

All but four patients, two of which had already reached the eighth, one the fourteenth, and one the thirty-fifth day of

disease respectively, were in the first week of their illness, the average time during which they had been ill being 3·92 days. It is interesting to compare with these figures the results of H. Mackenzie, who states that early disappearance of knee-jerks is extremely rare, except in fatal cases. Macdonnell (4), on the other hand, who pointed out that knee-jerks may be lost on the first day of the disease, did not attach any prognostic importance to their absence. As only four of my hundred cases proved fatal, the difference in my results is to be accounted for by the fact that Mackenzie wrote in pre-antitoxin times.

In the great majority of cases the reflexes which were lost did not return, nor did those which became sluggish under observation resume their normal activity during the time they were in hospital.

As a general rule, the severer the case the earlier the Achillis jerks were lost. In most cases, unlike what happens in tabes, they were affected after the knee-jerks. Undue briskness of the Achillis jerks, with or without a similar condition of the knee-jerks, was found in a few cases during the early stages of the disease. Inequality of the reflexes on the two sides was also observed in some cases. This was especially noted where the reflexes which had been lost returned while the case was still under observation. The Achillis jerk, for instance, might return and resume its normal activity on one side for some time before any response could be obtained on the other. In every case loss or impairment of the Achillis jerks outlasted any paralysis that might have been present. A similar condition was observed in the case of the knee-jerks by Berhardt (5), and also by Mackenzie many years ago, and more recently by Aubertin and Babonneix.

Summary.

(1) The tendo Achillis jerks are affected in a considerable proportion of all cases of diphtheria, though less frequently than the knee-jerks.

(2) The frequency and extent to which they are affected

bear, like albuminuria and paralysis, a direct relation to the character of the initial faucial attack.

(3) They are completely abolished in all cases of diphtheritic paraplegia.

(4) Their absence may be the only evidence of loss of motor power in the lower limbs.

(5) Like the knee-jerks, they are liable to be affected at an early stage of the disease, and to remain absent after disappearance of all diphtheritic paralysis, properly so-called.

(6) Like the knee-jerks, again, they may be unequally affected on the two sides, and, like the former, they may be unusually brisk before they become sluggish and finally disappear.

(7) The Achillis jerk, like the knee-jerk, after it has been lost, may reappear on one side before it does so on the other.

References.

(1) E. Bramwell. "Contribution to the Clinical Significance of the Absence of the Tendo-Achillis jerk," Brain, 1901, p. 554.—[Aubertin. *Archives générales de Médicine*, 1901, p. 330.—Aubertin and Babonneix. *Presse Médicale*, Fev. 8, 1905. Quoted by E. Bramwell, *op. cit.*]

(2) Mackenzie, H. *St. Thomas's Hospital Reports*, 1890.

(3) Rolleston, J. D. *Practitioner*, November and December, 1904.

(4) Macdonnell. "On the Early Loss of the Knee-jerk in Diphtheria," *Medical News*, October 15, 1887.

(5) Berhardt. *Virchow's Archiv.*, 1885. Band 99 S. 403.

Reviews.

Traité de Pathologie Mentale, publié sous la direction de M. Gilbert Ballet, par MM. D. Anglade, F. L. Arnaud, H. Colin, E. Dupré, A. Dutil, J. Roubinovitch, J. Séglas, and Ch. Vallon (pp. 1,600, 26 francs. Paris: O. Doin, 1903).

This large volume of 1,600 pages with 215 figures interpolated in the text, and six chromo-lithographic plates, is stated in the introduction to be a treatise on mental pathology. The authors hold the view that the mind is not an entity, and that if psychology has long considered it to be so, it is by virtue of an illusion which leads us to attribute to certain phenomena of consciousness an independence which their physiology does not justify: "aujourd'hui il ne peut plus être envisagé que comme une expression synthétique qui désigne l'ensemble des fonctions ressortissant à l'écorce du cerveau." Mental pathology is thus closely subordinated to the pathology of the cortex, and necessarily supposes an organic modification, innate or acquired, transitory or lasting of one or several of the constituent elements of the layers of the cortex. Mental pathology not constituting the entire pathology of the cortex, the question is discussed whether it is the pathology of any special region of the cortex. The views of Meynert, Flechsig, and others, are briefly discussed with the conclusion, "il est vraisemblable que la plus grande partie de l'écorce et peut-être toute l'écorce concourt, dans une certaine mesure, à l'exercice de l'intelligence: au moins actuellement on n'est pas en droit de dire qu'il n'en est pas ainsi." What is affirmed is that integrity of the cortex is the condition of integrity of the intelligence, and reciprocally that mental disorders presuppose a perturbation of one or several of the conditions regulating the normal functions of the cortex.

The discovery of the lesions of general paralysis, cellular changes which appear to condition certain deliria of infectious or toxic origin, and the relation of mental troubles to various toxæmias have lent new support to the doctrine which subordinates disorders

of the intelligence to material lesions or disorders of nutrition of the cortical cells.

With regard to classification, the authors remark that an imperfect science is only susceptible of imperfect classification, which is the case in mental pathology. Certain conditions, such as mania and *confusion mentale*, are syndromes of varying etiology and, therefore, cannot be classed in the same rank and on the same plan as affections constituting perfectly defined nosological individualities such as intermittent insanity or progressive general paralysis.

The work is divided into ten books. The first is concerned with the history, general etiology, diagnosis, and semeiology of mental disorders, and, for the historical section, Dr. Ballet is responsible.

The second chapter of the first book on the etiology of mental affections is by Dr. Anglade, who gives a comprehensive table dividing causes into: (I.) Le terrain psychopathique, and (II.) Les causes déterminantes des psychopathies.

The latter are subdivided into:—

(*a*) Biological and physiological (age and sex).

(*b*) Social, political, religious, intellectual and moral.

(*c*) Mechanical (concussion, trauma, compression, &c.).

(*d*) Physical (cold, heat, light).

(*e*) Pathological (affections of nervous system, of other organs, infections, and intoxications).

With regard to the influence of heredity in the causation of insanity, a brief summary is given of the conflicting views of Weissulann and others, as to the transmission of acquired peculiarities, and the opinion is held that what is transmitted, anatomically speaking, is an imperfect development of one or several central "territories," but with the saving clause that it is best to avow that we know nothing about the organic conditions of the *terrain psychopathologique*. Reference is made with disapproval to the attempt to differentiate a special group of cases to be known as *folies héréditaires*, as the term could not be applied specially and exclusively to certain forms of mental disorder.

The chapter on etiology occupies forty-seven pages, a summary being given of the present state of knowledge as to the causes of insanity, with many references to recent literature.

With regard to the influence of uterine and ovarian disease in the production of insanity in women, Dr. Anglade says: "Chez la femme, les lésions de l'utérus et de ses annexes ont été souvent incriminés pour expliquer la folie et on leur a fait jouer un rôle qu'on n'est véritablement pas en droit de leur attribuer."

Under the head of infections reference is made to the as yet inconclusive evidence of a microbic origin of "acute delirium," and to the effects of influenza, typhoid fever and malaria. He argues with Clouston as to the special liability of tubercular patients to suffer from suspicion and ideas of persecution, although other forms of mental disorder may occur in this connection. The importance of auto-intoxication is fully referred to with the statement that the nature of the poison is at present problematical.

Chapters III. and IV. of Book I. are by Dr. Séglas on the methods of examination of patients and the semeiology of mental diseases. The subject of measurement of the skull in various directions, and the calculation of cranial and facial indices is discussed at length, but the limitation of the value of these measurements in psychiatry is recognised. Five pages are devoted to deformities of the ears, which are not considered to be notably more frequent in the insane than the sane.

The present state of knowledge as to such questions as auto-intoxication, urotoxicity, and bacteriological examination of the blood, are fully considered.

The psychical symptoms of insanity are studied under the headings of (1) disorders of language (spoken and written); (2) the acts of the insane; (3) morbid states of cænesthesis; (4) states of intellectual enfeeblement and debility; (5) hallucinations; (6) delirious ideas (= Wahnideen and delusions).

Mercier's classification of delusions is given in full with approval.

Book II. contains two chapters only on mania and melancholia by Anglade and Ballet.

These conditions are described as "syndromes" of variable etiology and uncertain pathology, and cannot, therefore, be looked upon as definite diseases. It is worthy of note, however, that the authors do not relegate these time-honoured names to the past, but recognise that they still have a considerable clinical importance.

Book III. is connected with mental disorders in intoxications and infections. With the exception of the chapters on délire aigu and confusion mentale, which are written by Dr. Anglade, the whole of this section is from the pen of Dr. Roubinovitch. Psychical poisons are divided into exogenous (organic and inorganic), endogenous (diabetic, uræmic, hepatic, and thyroidal), and bacterial (infections.)

The chapter on alcoholism and the alcoholic mental disorders

is very complete, and many diagrams, illustrating the relationship of alcoholism and crime in different countries, are given. Great stress is laid on the "prophylaxis" of alcoholism by legislation and general education.

Separate chapters are devoted to délires fébriles, délires des septicémies, and délire du collapsus.

Dr. Roubinovitch limits the latter to mental disorders supervening in cases of sudden adynamia accompanying certain grave febrile affections, and differs in this respect from Kraepelin's description of Collaps-delirium.

Dr. Anglade devotes two chapters to délire aigu and confusion mentale. He considers the former equivalent to the delirium acutum of German and acute delirious mania of English writers, and that it is not merely a very acute form of mania nor, on the other hand, a febrile delirium. The observations of Bianchi and others as to a specific micro-organism in the blood in this condition are mentioned with the avowal that French alienists show little partiality for this view of its origin. Dr. Anglade, however, considers it to be of "nature infectieuse," as shown, not only by its symptomatology, but by the diffuse visceral lesions it produces.

"Confusion mentale" is defined as a syndrome which ordinarily develops primarily as the result of intoxications and infections, but which may also secondarily complicate other mental affections (mania, epilepsy, alcoholic delirium, and organic cerebral affections).

The following synonyms are given :—

Démence aiguë (Esquirol); stupidité, stupeur (Georget, Dagonet); confusion hallucinatoire (Delasiauve); torpeur cérébrale (Ball); hallucinatorischer Wahnsinn (Krafft-Ebing); amentia (Meynert, Serbsky); mania hallucinatoria (Mendel); acute confusional insanity (Conolly, Norman); paranoia dissociativa (Ziehen).

Dr. Anglade considers "confusion mentale" to be neither a pure vesania nor a simple state of intoxication or exhaustion, but a combination of psycho-neurosis and cerebral infection. He groups many post-operation, puerperal and post-febrile cases under this group, and describes two principal forms: (1) stupidité, or confusion mentale asthenique, and (2) délire hallucinatoire aigu, or confusion mentale délirante hallucinatoire.

Book IV., on "psychoses constitutionelles," is entirely written by Dr. Arnaud, and consists of three parts :—

(1) Psychoses chez les sujets à prédisposition latente.

(2) Psychoses chez les sujets à prédisposition apparente.

(3) Démence vésanique.

Part I. contains as its principal subject a description of "délires systematisés ou partiels," the equivalent of Paranoia or Verrücktheit of German writers. A most comprehensive summary is given of the views of writers in different countries as to this much-debated subject, and a most valuable table of German classification of Paranoia and French equivalents. Dr. Arnaud admits of an acute and curable, as well as a chronic and incurable, group of cases of this nature. The whole subject is treated at great length and with complete thoroughness.

The second chapter of this part is on "psychoses périodiques ou intermittentes," and is well illustrated by charts of the course of such cases.

Part II. contains the consideration of manie raisonnante, folie morale, persécutés-persécuteurs, délire systématisé originel, obsessions (including sexual anomalies and perversions), exaltation maniaque et mélancholie raisonnante. The author abandons the term "dégénérés" for individuals of this group, and prefers the expression "déséquilibrés" as not implying any doctrinal notion.

Part III. discusses démence vésanique tardive and démence vésanique précoce. The question of "dementia præcox" is fully considered. With regard to Kraepelin's inclusion of paranoid forms in dementia præcox, Dr. Arnaud says: "Nous continuons à penser avec tous les observateurs français que la démence précoce et les délires systématisés dans leur ensemble doivent être séparés dans les classifications aussi nettement qu'ils se distinguent en clinique, et que la dénomination de *démence paranoïde* doit être limitée à certain cas de démence précoce qui débutent par une phase de délire vaguement systématisé."

Book V. treats of the neuroses considered only from the point of view of the mental disorders occurring in them, the first chapter on the "Etat mentale des hystériques," being written by Dr. Colin, and the remainder on mental disorders in epilepsy, psychical epilepsy, chorea (both Sydenham's and Huntington's chorea), and Parkinson's disease by Dr. Dutil.

Dr. Colin describes the short delirious manifestations of ordinary hysteria, as well as somnambulism and ambulatory automatism. He does not believe in the existence of a special "hysterical insanity" or "hysterical mania," but merely in hysteria associated with insanity of any clinical variety.

The psychical manifestations of epilepsy are described by Dr. Dutil under the following headings:—

(1) Les syndromes avec excitation.
(2) Les syndromes avec depression.
(3) Les formes alternantes, and (4) epileptic dementia.

Dr. Dutil does not admit the existence of a special "choreic insanity," but agrees with Jeffrey that insanity associated with chorea is a manifestation of degeneration.

Book VI. is perhaps the most important in this work, and is entirely written by Dr. Dupré. It is entitled "Psychopathies organiques," and contains chapters on progressive general paralysis, organic encephalopathies (hæmorrhage, softening, meningitis, abscess, tumour, scleroses, syringomyelia, myopathies, &c.), and organic dementia.

The chapter on general paralysis is very complete and profusely illustrated, both in the clinical and pathological sections. The author agrees with Sérieux and Mignot that a "sensorial," variety of general paralysis exists, characterised by the predominence of aphasia, hallucinations, hemianopsia, &c., and in which the lesions predominate in the middle and posterior part of the cortex. He believes that in certain cases true general paralysis may end in recovery, and considers it illogical to regard these as instances of pseudo-general paralysis.

Under the head of diagnosis he lays great stress on the value of lumbar puncture, the discovery in the fluid thus withdrawn of small lymphocytes staining deeply being almost universal in cases of general paralysis and preceding any motor disorders.

The presence of lymphocytes, however, merely settles the question of the organic or inorganic nature of the case as the same condition is met with in tabes, chronic or subacute meningomyelitis, syphilitic or not, tubercular meningitis, and chronic alcoholic meningitis. He sums up as follows: "Le résultat positif du cyto-diagnostic cephalorachidien n'indique l'existence de la paralysie générale que lorsqu'il n'existe aucune raison, étiologique ou clinique, d'imputer à un autre processus méningitique la lymphocytose constatée."

A very full description is given of Korsakoff's polyneuritic psychosis, which is considered to be an attenuated expression while délire aigu is an intense expression of intoxication of the nervous system in the predisposed. The pathological condition is very fully considered with the conclusion that, "l'affection psychopolyneuritique apparaît comme consécutive à l'imprégnation de l'ensemble du systême nerveux par les poisons en circulation dans l'organisme malade." Under brain tumours, a

table is given of the relative frequency of psychical disorders in association with tumours of different areas of the brain, the highest proportion being shown in tumours of the corpus callosum, and next of the frontal region.

Book VII., by Roubinovitch, treats of mental disorders in congenital or infantile cerebral lesions (idiocy and imbecility), and is as complete and well illustrated as the rest of this work.

Dr. Roubinovitch is also responsible for Chapter I. of Book VIII., treating of myxœdema and cretinism, while Dr. Dutil discourses on mental disorders from over-function of the thyroid body (exophthalmic goitre).

Dr. Anglade writes fully in Book IX. of the treatment of mental disorders, and Book X., which completes the work, is by Dr. Vallon on administration, lunacy law, asylums, experts, civil capacity, responsibilities, crimes, life insurance, traumas, feigned insanity, and deontology.

There appears to be only one adverse criticism to make on this monumental work, which is of encyclopædic importance, and redounds in the highest degree to the credit of our French colleagues, and must ever remain a most valuable work of reference.

The criticism is directed against the index, which is merely a table of contents of the different chapters. For such a large work a full index should have been given so as to facilitate the labour of searching through the different chapters for any special subject to which reference is desired. Considering the size and weight of the book, it would have been better in two volumes, but its extraordinarily low price (26 fr.) is an inducement to everyone engaged in psychiatry to purchase it.

R. Percy Smith.

Myoclonus Multiplex. By Charles L. Dana. (*Journal of Nervous and Mental Disease*, August, 1903).

Pathology of Paramyoclonus Multiplex (Friedreichs Type). By J. Ramsay Hunt. (*Journal of Nervous and Mental Disease*, July, 1903).

Die Progressive Myoklonus-Epilepsia (*Unverricht's Myoklonie*). By Dr. Hermann Lundborg.

The above recent monographs on myoclonus may well be reviewed together, for they raise the important question as to what is meant by the name "myoclonus." Is "myoclonus" a

disease—an entity—or is it only a symptom? As originally used by Friedreich, it certainly indicates a disease having definite clinical symptoms. Unverricht attached the name to a class of cases differing entirely from that described by Friedreich. How, then, should the term "myoclonus" be used?

Dana starts by the statement that myoclonus is a general term covering all the diseases in which muscle-twitching is a prominent symptom. The term here is used as indicating a symptom, and as such he proceeds to classify the diseases in which the symptom occurs; the classification he arrives at is as follows:—

(1) Paramyoclonus multiplex, of Friedreich; astasic myoclonia, of Vanlair; multiple spinal myoclonus, of Lowenfeld; fibrillary chorea, of Morvan; fibrillary myoclonus, of Kny.

(2) Functional or hysterical myoclonus multiplex, chorea major, chorea electrica, of Henoch.

(3) Myospasmia; memory-spasms, of Friedreich; habit chorea; chorea variable des degenerès; convulsive or spasmodic tic; tic general; Tourette's disease, myriadrit, palmus; tic neurosis, of Collins.

(4) Degenerative chorea, hereditary chorea, Huntingdon's chorea; myoclonus-epilepsy; myoclonus, of Unverricht, of "familial" type; myoclonia congenita, of Seeligmüller (?); hereditary degenerative chorea of Sachs.

(5) Infectious chorea, chorea minor, Sydenham's chorea; chorea electrica, of Dubini (?); chorea electrica, of Bergeron; senile chorea, of Gowers (?).

It is difficult to see what advantage can be derived from a classification which puts under one head diseases which are essentially different in type.

The "tics" form a most useful clinical group, and, as a type, are easily distinguished from Sydenham's chorea. If the term "myoclonus" is to include all diseases in which spasmodic muscular movements occur, which neither clinically nor pathologically —so far as pathology goes—have anything in common, then the term loses its significance and has but little value.

It would be well that the term "paramyoclonus multiplex" or "myoclonus multiplex" should be reserved for that form of myospasms characterised by multiple isolated contraction of individual muscles.

Hunt goes still further, and would distinguish contractions of this type from the contractions of a cerebral type which are characterised by movements of a more or less co-ordinate nature.

It would be well if the term "myoclonus" could be reserved for the two conditions above mentioned, viz., the paramyoclonus of Friedreich and the myoclonus of Unverricht.

Hunt has had the opportunity of investigating pathologically one case of myoclonus of Friedreich type. He finds no abnormality of the nervous system, except some slight change in the vessels of the gray matter of the spinal cord. The muscles, on transverse section, have a diameter of unusual size, measuring in some instances 175 micromillimetres. In contrast to this are very small fibres having crescentic forms and flattened out on the periphery of the large fibres. Instead of finding the nuclei of the sarcolemma sheath confined to the periphery of the muscle fibre and just beneath this membrane, they are found scattered as well between the sarcous elements. Sometimes three or four nuclei occupied a central position in a single fibre. The nuclei of the sarcolemma sheath are increased in number.

Lundborg, in an excellent monograph on *Die Progressive Myoklonus-Epilepsia*, divides the myoclonias as follows:—

(1) Myoklonia symptomatica.

(2) Myoklonia simplex, or myoklonus multiplex.

(3) Myoklonus-epilepsia, divided into two forms: (*a*) a progressive form (Unverricht's family type); (*b*) an intermittent sporadic form.

The tics, Huntingdon's chorea, should not be confused with the myoclonia, from which they are easily distinguished.

This classification would commend itself to the writer of this review, not only on account of its simplicity, but also because it allows of a wide use of the term as a symptom in association with other diseases, and recognises two well-marked types of disease which may be considered as entities.

The monograph deals more especially with the one form of myoclonus epilepsy, and the following description of the disease is given:—

The disease begins in later childhood and lasts during life, so that some of the patients between 60 and 70 years old have suffered from fifty to sixty years.

Most patients, however, die about middle age. Three stages are described: the first, characterised as the epileptic-tetaniform stage, is of comparatively short duration. The attacks occur mostly at night and during sleep, at first with a long interval, but always tending to become more frequent.

The patient awakes out of sleep with painful tonic or clonic and tonic cramp in various parts of the body. The attacks are

very different in their intensity. In the mildest attacks rapid muscular contraction takes place in the extremities. In the severest cases the attack resembles tetanus, and the contractions follow one another so quickly that the muscles never become flaccid. These contractions do not produce a locomotor effect, owing to the fact that the antagonistic muscles are in like contraction. Every manipulation tends to increase the spasm. Consciousness is preserved, and the patient has pain and distress, and may cry out with pain. Urine and fæces are not passed incontinently.

The second stage, so-called myoclonic epileptiform stage, lasts for several years (ten or more). The disease is now more pronounced during the day. Tremor — fibrillary and fascicular muscular contractions occur — beginning usually in the upper extremities, then in the buttock muscles, the throat, the face, and, lastly, the diaphragm, the pharynx and larynx, and, finally, the ocular muscles and sphincters of the bladder and rectus—in a word, all the voluntary muscles.

The patient experiences good and bad days.

During this stage psychical phenomena present themselves and the intelligence falls off; the patients exhibit changes between depression and exaltation.

The third—terminal—stage is one of cachexia. This may begin at almost any time, but generally shows itself after the first decade of the disease.

The muscular movements gradually increase in the course of years, whilst the epileptic attacks become less frequent, and eventually disappear. The periodicity of the attack becomes irregular, and the intermediate periods of rest shorter.

Rigidity of muscles now manifests itself, and the patient assumes a fixed and bent position. Any attempt to alter the position produces a definite resistance. The patient sweats and salivates profusely, passes into a somnolent half-stupor stage. Urine and fæces are often passed incontinently; the cachexia increases, and the patient dies of inanition or of some complication.

Two factors in the etiology of the disease came into prominence; the first of these is the hereditary and family nature of the disease, and secondly the influence of alcohol.

In two families there were five cases; in one family four cases; in seven families three cases; and in nine families two cases.

The occurrence of paralysis agitans in other members of the

same family is a striking feature brought out in the cases recorded, for in one family in which five cases of myoclonic epilepsy occurred five members of the family were affected with paralysis agitans.

The author next deals with the differential diagnosis of the disease.

The important diagnostic points are :—

It is a family disease—starting with nocturnal epilepsy—associated in childhood with attacks resembling tetanus, and after a few years lightning-like involuntary muscular contractions which gradually affect all voluntary muscles. The patient has good and bad days. The muscular contraction frequently affects only a part of a muscle. The contractions occur irregularly and are increased by voluntary movement; during sleep they stop or are very much diminished. As time goes on a definite muscular rigidity occurs. The knee-jerks are exaggerated. No trophic disturbance of muscles or abnormality of sensation is present. Sweating and salivation are marked features. After some years failure of mental powers, sometimes associated with mania and hallucinations occur.

The author next contrasts the disease with chronic progressive chorea (Huntingdon's chorea). Both diseases arise from a degenerative basis, have a progressive course, and end in dementia. The involuntary contraction of muscles is present in both cases.

The points of distinction are :—

In Progressive Myoclonus Epilepsy.	*Huntingdon's Chorea.*
(1) Direct heredity unusual.	(1) Direct heredity usual.
(2) Disease begins in childhood, with nocturnal epilepsy—and later on the muscular contractions appear—the muscular contraction and the attacks stand in relation to one another.	(2) The disease affects persons between 30 to 40 years of age and is rarely combined with epilepsy.
(3) Periodicity dependent on various sources of reflex irritation.	(3) No periodicity.
(4) Sensory clonic reaction present.	(4) Sensory clonic reaction absent.
(5) The muscle contractions are myoclonic— Sharp contractions affecting single muscles.	(5) The muscle contractions are choreic— Slow contractions affecting groups of muscles.
(6) On intention movement the contractions increase.	(6) On intention movement the chorea becomes less.
(7) The patient soon loses the power of walking.	(7) The power of walking, though difficult, is not lost till the final stage of the disease.

The differential diagnosis between this disease and Koschewnikow's epilepsy and Dubini's chorea electrica is then

discussed, and the similarity of myotonia (Thomsen's disease) to myoclonia is pointed out.

Pathological Anatomy.

In the few cases which have been examined, the changes found have been slight and often attributable to some intercurrent condition.

In Clark and Prout's cases changes were found in the second layer of cells of the cortex, and also in the large pyramidal cells of the third layer—and these authors suggest that these changes underlie the myoclonic spasm.

It would seem, therefore, probable that myoclonus is dependent on changes in the Rolandic region of the cortex.

The possible relation of the disease to changes in the thyroid and parathyroid is discussed, for it has been shown experimentally that clonic spasms occur after removal of the thyroid.

Ramsay Hunt, in the examination of a case of paramyoclonus multiplex, which would come under Lundborg's second division, could find no change in the nervous system, but found changes in the muscles, the fibres being of an unusually large size—with an increase of nuclei in the muscle fibre and also in the sheath of the muscles.

In conclusion of this short review, it would seem that but little advantage is gained by placing under the term myoclonus all the various conditions enumerated by Dana, and it would be far better to adopt the classification given by Lundborg:—

(1) Myoklonia symptomatica—using the term myoclonus simply as a symptom indicating a form of myospasm characterised by multiple spontaneous, and isolated contraction of individual muscles.

(2) Myoklonus multiplex.

(3) Myoclonus epilepsy—(*a*) A progressive form. (*b*) An intermittent form.

Frederick E. Batten.

Isolement et Psychothérapie. Traitement de l'Hystérie et de la Neurasthénie. Pratique de la ré-éducation Morale et Physique. By Drs. JEAN CAMUS et PHILIPPE PAGNIEZ. Préface du Professeur J. DEJERINE. (Paris: Félix Alcan. 1904. Pp. 407.)

THE object of this book is to demonstrate the method and the results that accrue in the treatment of hysteria and neurasthenia, as it is now carried out at the Saltpêtrière in the wards of Prof. Dejerine. In a short but interesting preface Prof. Dejerine remarks on the neglect of moral force by medical men in their treatment of nervous diseases; he considers that although moral influence is at last beginning to be more adequately recognised as a power in therapeutics, it is still far from sufficiently appreciated. Finding that the results obtained by the use of the various factors that make up the Weir-Mitchell treatment did not come up to his expectations, he was gradually led to see that this treatment should be regarded only as a means to bring the patient under moral influence, that is, in the current phraseology, as a means to the use of psychotherapeutics. Further, the inconveniences, and often bad results that arise from the treatment of hysterical patients in a general ward of a hospital and from the effect upon them of the presence of other patients similarly affected, led him to devise a plan by which poor patients in hospital could have the same advantages as the rich in private nursing homes. This he accomplishes by isolating each bed by means of curtains, and since he has adopted this measure the results of hospital treatment have been as good as those obtained in nursing homes in patients of the well-to-do classes.

The authors have carried out Prof. Dejerine's mode of treatment under his supervision, and are thoroughly conversant with it. In this book they attempt the somewhat difficult task of describing all the details of the method. Obviously where so much depends on the individuality of the physician, and on the idiosyncrasies of each patient, and where the treatment rests upon such an undefined and intangible quantity, as the moral influence of the one upon the other, any description which can present all the points to a reader without obscurity is a matter of great difficulty. Again, the very secret of success eludes description, because it lies in the physician's ability to seize upon the higher moral qualities of his patient, and to enlist them into his service to overcome the lower tendencies which have resulted in nervous disorder. To their

general account of the method, the authors have, therefore, wisely added the description in detail of a number of cases. These cases are well reported, and each ends with some short critical remarks on the character of the illness, and the result of the treatment. More than one-third of the book is taken up by these case reports. The first part of the book deals historically with the subjects of isolation, of "faith cures" by various means, of hypnotism and suggestion, and of psychotherapy by "persuasion." These several topics are treated in the widest possible sense, and the historical *résumé* given cannot fail to be of interest. Under isolation the authors discuss hermits, religious retreats, life in monasteries and convents, and allude to a now-forgotten but former popular author, Zimmermann, of whom O. W. Holmes said that he was the only man to whom he would intrust his latch-key. "Suggestion" is carefully distinguished in this book, as in that of Prof. Dubois (of Berne), from "persuasion"; the former is condemned, and is to be superseded by the latter. The distinction though clear in theory, in practice would seem to become rather fine, especially from "suggestion in the waking state." Under the history of persuasion there is included a short but interesting account of that form employed by the Roman Church as "spiritual direction." The second part of the book is that which embodies the essential teaching of the authors. In it they discuss fully the best means of carrying out isolation of nervous patients under varying circumstances, the rules for rest and dietetics, and for the employment of massage and electricity.

A chapter on the influence of the mind on the body, and on the reverse effects of the body on the mental state, leads to a discussion on hypnotism, which is rejected as a therapeutic agent, partly on account of the difficulties and dangers inseparable from its employment, partly because other less objectionable measures can be substituted for it with good results, the method of persuasion being, as said above, employed instead of suggestion.

The manner of carrying out this method by the physician is discussed in full detail, and instructions are even given as to what he should do and say during "entretiens psychothérapiques"; a considerable part of psychotherapy lies in successfully impressing upon the patient that he or she is suffering from a curable affection, and, afterwards, "persuading" them of the improvement that is taking place in their condition. The authors, however do not tell us what we should do with that very troublesome person with whom we sometimes meet, who is never tired of insisting that he is no better, nor with that neurasthenic who is

not disposed to take anything on trust, but is prepared to argue about treatment with his doctor to an unlimited extent. It further seems to follow that successful "psychotherapy" almost demands as a *sine quâ non* that the doctor should be the intellectual superior of his patient. This is not always the case.

A passing allusion to a useful chapter on the re-education of hysterical and neurasthenic patients, that is the restoration of their physical, moral, and intellectual powers to the normal standard of each, that they may again take their places as normal members of society, and of the means to be employed in this difficult task, may conclude this notice of a book which is well written, highly interesting, and deals with the beginnings of a rational mode of treatment which is likely to proceed to further development in the future.

J. MICHELL CLARKE.

On Unilateral Hallucinations of Hearing. By E. LUGARO (Sulle Allucinazioni Unilaterali di Audito). *Rivista di Patologica Nervosa e Mentale*, May, 1904.

Dr. LUGARO reports a case of hallucinations of hearing assigned to one ear. The patient was 39 years old, a smith by trade, had an aunt and one sister insane. In February, 1898, after the death of his father, he fell into a deep melancholy. He tried to kill himself with a small revolver, the charge of which burst the tympanum and lodged in the temporal bone. Most of the contents were extracted; probably some fragments remained, for suppuration continued. He was sent to the asylum, where he improved so quickly that he was discharged after twenty-four days, committed to the charge of his wife. He returned to his work. Two years and six months after, his wife meeting with an accident, he talked of suicide, became maniacal, and was again brought to the asylum. He was motionless, silent, and passive, refused food, and did not sleep. He said that the voice of his dead father was ever in his right ear, repeating "suicidati," but he resisted the suggestion. He said that for two years back there was a continuous noise in his ear, like the cry of a cricket, but low and uniform. In a month he was well again and able to leave the asylum. The sound in the ear continued, but did not take the form of words. After a time the hallucinations of hearing returned. Then he began to hear what he had been thinking repeated into his right

ear. In the course of two years the hallucinations became more frequent, returning almost every day. He fell into a state of melancholia attonita, and was again brought to the asylum. He was thin and anæmic; the right tympanum was found to be loose, but cicatrised. Hearing gone on the right side, but there was unusual excitability to the galvanic current. The patient was soon again able to go home and return to his usual work. Dr. Lugaro can only find two other examples of the hallucinations of voice repeating the thoughts. In this case we have a peripheral irritation and a morbid state of the brain. Unilateral auditory hallucinations, however, have been described by many observers. In some cases it looks as if the affection of the ear was the cause of the insanity, though we may assume a morbidly excitable condition of the brain. The peripheral lesion favours the production of hallucinations though not a sufficient cause, without an abnormal activity of the brain. Mabille has described a case in which the hallucinations ceased after the ear disease had been cured. Lugaro tells us that all the insane who are deaf are affected with auditory hallucinations. Lunatics who are blind may still have visual hallucinations.

WILLIAM W. IRELAND.

Die Färbetechnik für das Nervensystem. By BERNHARD POLLACK. (Third Edition. Berlin, 1905. S. Karger. Price 3s. 6d. net.)

DR. POLLACK'S compendium is arranged as follows: (1) The removal of the brain, its naked-eye examination, and its preparation for museum purposes; (2) hardening fluids and microscopic technique; (3) the influence of preservatives on brain weight, micrography and photography (a chapter of three pages only); (4) methods of staining the three chief constituents of nervous tissue; (5) general remarks.

In his chapter on methods of hardening, the author emphasises the axiom, the better the fixation the better the microscopic specimen. The postulates of an ideal fixative are these: it shall fix without shrinking; it shall serve as an imbedding medium, and so do away with the necessity for dehydration and clarification; it shall act rapidly, but not cause brittleness when immersion is prolonged, and it shall be neutral in the presence of different stains, so that sections from one block may be variously treated

and display all constituents. But, of course, we are still far off our ideal. Pyridin, which, by the way, does not occur in Dr. Pollack's list, comes nearest, for it combines the properties of a fixer, a clarifier, and a dehydrator, but it has disadvantages in causing shrinkage and in requiring paraffin imbedding, not to mention its offensive odour. In permitting the use of many methods, formalin almost satisfies one postulate, and it rightly occupies the place of honour in this compendium.

In the series of staining procedures we observe none that has not been described elsewhere, but the running commentary—an important feature of the work throughout—is full of sound suggestions, and proves the writer to be a thoughtful, accomplished, and well-practised microscopist. After Weigert, it is insisted that a perfect stain should be selective, certain, time-saving, intense in colouring, and permanent; that, for the sake of orientation, it should make other objects in the field visible, and that it should not require drastic, intercurrent treatment.

We know too well that it is impossible for any worker single-handed to test the merits of the ever-increasing multitude of neuro-histological methods; therefore, rather as co-operators than as critics, we will indicate some omissions and shortcomings which we have noticed, and which, we hope, will receive attention in future editions. The method of Marchi in its original form is obsolete; as workers at Queen's Square have demonstrated, when it is desired to follow a degenerated tract of nerve fibres, hardening in formalin and the use of Busch's osmic acid and iodate of sodium mixture are much more advantageous; while, if the tissue be hardened in Müller's fluid, we follow Dr. Orr, and assist the osmic penetration with acetic acid. Again, if we wish to display normal nerve fibres in formalin-hardened tissues, we find in the solutions of fluorchrome and of chrome alum, used by the late Professor Weigert for other purposes, excellent mordants for myelin. The mixture of perchloride of mercury and picric acid which finds favour as a fixative among Italian histologists, Unna's polychrome blue as a stain for nerve cells, the platinum method of Dr. Ford Robertson for displaying neuroglia, and the fresh method of Dr. Bevan Lewis, all deserve notice; and, finally, the remarks on micrography should be either extended or cut out.

Apart from these imperfections, the compendium will prove a useful companion to the more comprehensive work of Bolles Lee. The author's discrimination between important and unimportant methods is called for, and we welcome the complete list of references appended to each chapter.

ALFRED W. CAMPBELL.

The Anatomy of the Brain. (A Study of the Human Brain from the Brain of the Sheep.) By J. F. BURKHOLDER, M.D., Professor of Anatomy in the Illinois Medical College. With an introduction by Professor HENRY H. DONALDSON, of Chicago. (175 pages, octavo, 32 full page plates [5 coloured]. Cloth. Price, 2 dols. post paid. Chicago: G. P. Engelhard & Co.).

AT times when human material was unprocurable for teaching purposes, Professor Burkholder tested and proved the usefulness of the brain of the sheep, and he now issues a guide to its dissection.

The manual will be welcomed by the student of medicine, and even more so by the graduate, for it will enable them, independently and privately, to acquire familiarity with, and renovate, their knowledge of an anatomical "part" as difficult as it is important.

The various dissections are carefully planned, the illustrations well reproduced, and, the descriptions, on the whole, correct and commendably brief; but the attention of the reader is too frequently arrested by confusion of tenses, by loose construction, and by misprints, all of which should have been eliminated before the "revise" was approved. It is also to be regretted that the author has intentionally omitted references to homologies, for much that was previously obscure in this direction has been removed by the recent researches of Elliot Smith, and others, and surely the interest of the student would be accentuated if he were told the human equivalents of the various gyri and sulci coming under inspection.

The English tongue may trip over the expression "hemisphærium cerebri," still it is pleasing to find Professor Burkholder adopting the nomenclature recommended by the Basle convention.

ALFRED W. CAMPBELL.

Essai sur les Alterations du Cortex dans les Meningites Aigues. By PIERRE THOMAS. (Paris, J. B. Bailliere et Fils, 19, Rue Hautefeuille.)

IN this work the author maintains that in acute meningitis of specific infective origin, all the layers of the cortex are affected. The first elements to show any lesion are the larger and more highly specialised cortical cells. These show alterations before any change can be detected in the vessels, and with an intensity which appears to be in relation to the infectivity of the cause, the duration of the infection and, apparently, the size of the elements themselves. The stages of such changes are, first, alteration in position, then of the shape, of the nucleus, pericellular infiltration, intra-cellular infiltration, and, lastly, destruction of the element. Judging from the cases observed by the author, neuroglial changes are secondary and appear slowly. The symptoms due to these lesions, be they exaggeration or diminution of neuron function, are delirium, motor symptoms, disorders of speech, coma, and alteration of sensation. In the opinion of the author, the lesions which he describes begin in the gray matter, and extend to the vessels and meninges. He believes that the *rôle* of the vessels has been considerably exaggerated, if not misunderstood. In other words, he regards the presence of specific diseases, such as pneumonia and tubercle, as having a selective effect on the large and other pyramidal cells of the cortex.

DOUGLAS STANLEY.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

AT the Clinical Meeting held at 11, Chandos Street, on Thursday, March 30, at 8.30 p.m., the following cases were shown :—

Dr. ORMEROD.—(*a*) *Case resembling Friedreich's disease, but exhibiting extensive anæsthesia.*

[1] Male, aged 21, formerly a clerk.

Family history.—Eldest of four children, of whom the next in order, viz., a sister, aged 19, is said to be similarly affected.

History of illness.—Weak on his legs from an early stage. Upper limbs affected (difficulty in writing first noticed) for the last five years. Speech affected, and affection of limbs worse since an illness (said to be "phthisis") three years ago. Numbness and formication in limbs, two years. Loss of sensation of passing urine one year.

Condition on admission to the National Hospital (January 20, 1905).—Unsteady gait. Hardly able to walk without touching neighbouring objects. Romberg's symptom present. Unable to write more than a few words at a time (painful cramp sets in). Speech somewhat slurring and nasal. Knee-jerks absent. Pupils normal. Anæsthesia to touch and prick as shown in charts. Loss of taste in anterior two-thirds of tongue. No loss of thermal sense, but a bath at 100° produces sensation of scalding and formication.

(*b*) *Case for diagnosis, (possibly) progressive disease of pons and peduncles.*

[2] Man, aged 28, formerly a country policeman, previous health good, no history of syphilis.

In May, 1903, sudden attack, in which eyes were drawn to left, and face drawn to left (lesion in neighbourhood of right sixth nucleus?), numbness left hand, giddiness and reeling to left.

Recovery in ten weeks; during recovery, diplopia, right eye turning inwards.

During the next ten months four further attacks followed, of

diplopia associated with right facial palsy, all getting well, though his walking appears to have remained unsteady. The sight in the right eye has been bad since the first attack; for this there is a local cause, a large veil opacity in the vitreous.

February, 1904.—Attack in which left leg became weak, and left arm weak and shaky (but no return of diplopia or facial palsy). After this attack the change in his voice developed.

On admission to the National Hospital (August 5, 1904).—Ocular movements fair (? some weakness right external rectus). Some weakness right lower face (which passed off). Speech rather husky and squeaky. Weakness, tremors, inco-ordination in movements of left limbs. Tendency to fall to left. Some rigidity left leg; knee-jerk increased left side. Left ankle clonus, and plantar reflex extensor. Numbness left limbs, but no objective affection of sensation.

August 29.—Paralysis in district of *left third nerve trunk*, which passed off gradually.

September 23.—Vomiting and giddiness; cannot stand alone; falls to *right*. Position of head = occiput drawn down towards right shoulder and chin rotated to left. This passed off.

November. — Rhythmical oscillations of palate and vocal cords (and diaphragm ?) first observed.

December 26.—Vomiting and gastro-intestinal disturbances ascribed to Christmas dinner, but followed by :—

December 28.—A *second* attack of paralysis in the district of the *left third* nerve trunk.

Main features at present time (March 17, 1905) are :—Left hemiplegia incomplete. Irregular movements of left arm, which are, however, less marked than formerly. Incomplete ocular palsies = deficient movement of convergence; deficient upward movement, left. Peculiar explosive and squeaky speech; this has become more marked since admission. "Nystagmus" of palate and vocal cords.

Dr. Gordon Holmes.—*Case of midbrain lesion with unilateral tremor.*

[3] A. V., aged 10 years, was admitted to the National Hospital in February, 1905.

Family history unimportant, previous health good.

Four years ago he became unsteady on his feet, and began to stagger like a drunken man. Gait has since steadily become worse. Has walked over to the left. Fifteen months ago his left arm was noticed to be weak and awkward, and a few months later the right arm began to shake.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

AT the Clinical Meeting held at 11, Chandos Street, on Thursday, March 30, at 8.30 p.m., the following cases were shown :—

Dr. ORMEROD.—(*a*) *Case resembling Friedreich's disease, but exhibiting extensive anæsthesia.*

[1] Male, aged 21, formerly a clerk.

Family history.—Eldest of four children, of whom the next in order, viz., a sister, aged 19, is said to be similarly affected.

History of illness.—Weak on his legs from an early stage. Upper limbs affected (difficulty in writing first noticed) for the last five years. Speech affected, and affection of limbs worse since an illness (said to be "phthisis") three years ago. Numbness and formication in limbs, two years. Loss of sensation of passing urine one year.

Condition on admission to the National Hospital (January 20, 1905).—Unsteady gait. Hardly able to walk without touching neighbouring objects. Romberg's symptom present. Unable to write more than a few words at a time (painful cramp sets in). Speech somewhat slurring and nasal. Knee-jerks absent. Pupils normal. Anæsthesia to touch and prick as shown in charts. Loss of taste in anterior two-thirds of tongue. No loss of thermal sense, but a bath at 100° produces sensation of scalding and formication.

(*b*) *Case for diagnosis,* (*possibly*) *progressive disease of pons and peduncles.*

[2] Man, aged 28, formerly a country policeman, previous health good, no history of syphilis.

In May, 1903, sudden attack, in which eyes were drawn to left, and face drawn to left (lesion in neighbourhood of right sixth nucleus?), numbness left hand, giddiness and reeling to left.

Recovery in ten weeks; during recovery, diplopia, right eye turning inwards.

During the next ten months four further attacks followed, of

diplopia associated with right facial palsy, all getting well, though his walking appears to have remained unsteady. The sight in the right eye has been bad since the first attack; for this there is a local cause, a large veil opacity in the vitreous.

February, 1904.—Attack in which left leg became weak, and left arm weak and shaky (but no return of diplopia or facial palsy). After this attack the change in his voice developed.

On admission to the National Hospital (August 5, 1904).—Ocular movements fair (? some weakness right external rectus). Some weakness right lower face (which passed off). Speech rather husky and squeaky. Weakness, tremors, inco-ordination in movements of left limbs. Tendency to fall to left. Some rigidity left leg; knee-jerk increased left side. Left ankle clonus, and plantar reflex extensor. Numbness left limbs, but no objective affection of sensation.

August 29.—Paralysis in district of *left third nerve trunk*, which passed off gradually.

September 23.—Vomiting and giddiness; cannot stand alone; falls to *right*. Position of head = occiput drawn down towards right shoulder and chin rotated to left. This passed off.

November. — Rhythmical oscillations of palate and vocal cords (and diaphragm ?) first observed.

December 26.—Vomiting and gastro-intestinal disturbances ascribed to Christmas dinner, but followed by :—

December 28.—A *second* attack of paralysis in the district of the *left third* nerve trunk.

Main features at present time (March 17, 1905) are :—Left hemiplegia incomplete. Irregular movements of left arm, which are, however, less marked than formerly. Incomplete ocular palsies = deficient movement of convergence; deficient upward movement, left. Peculiar explosive and squeaky speech; this has become more marked since admission. "Nystagmus" of palate and vocal cords.

Dr. Gordon Holmes.—*Case of midbrain lesion with unilateral tremor.*

[3] A. V., aged 10 years, was admitted to the National Hospital in February, 1905.

Family history unimportant, previous health good.

Four years ago he became unsteady on his feet, and began to stagger like a drunken man. Gait has since steadily become worse. Has walked over to the left. Fifteen months ago his left arm was noticed to be weak and awkward, and a few months later the right arm began to shake.

Four years ago he also developed squint; the left eye turned outwards, and since then he has not been able to look up properly. During his illness frequent headache and vomiting, and his mental condition has deteriorated.

Sight and hearing normal, no ophthalmoscopic changes. Paralysis of upward movement of the eyes, weakness of their downward movements and of left internal rectus. Pupils almost inactive to light, but contract on accommodation. Head and eyes generally deviated to left. Tremor of right half of tongue. Irregular tremor of head, which is generally rotatory.

No wasting of muscles; slight rigidity of right limbs, but power of both right and left fair. Except when they are fully supported, and during sleep, there is constant tremor of the right limbs involving both distal and proximal muscles. It is fairly regular in rate and range, but intermittent and often rhythmical. The oscillations are somewhat coarse, and about four to five per second. Increased by excitement and on effort, and cannot be inhibited by patient. Intention tremor on volitional movement. There is a tendency to athetoid movements of left hand, but no ataxia. His gait is very ungainly, reeling and staggering.

There is no sensory change. The tendon reflexes are brisk, and the plantars of the extensor type.

There is considerable mental defect.

Dr. Wilfrid Harris.—*A case of chronic chorea.*

[4] An engine driver, aged 63, while at work ten years ago strained his back, causing such pain in the left side of his back that he had to walk home doubled up. He has never walked properly since, and soon afterwards became unsteady in his gait. At the same time, though a teetotaller, his articulation became altered and slow, and he was often accused of being drunk. He has not been able to do any work for the last nine years.

There has been precipitancy of micturition, with occasional incontinence, for the last four years. His memory for recent events is not very good, and he has occasional difficulty in remembering where he is on waking. He knows of no other member of the family similarly affected, and he denies venereal disease.

There are now constant choreic movements of the face, head and limbs. The articulation is difficult to understand on account of its explosive character. The gait is markedly ataxic, and somewhat spastic. The grip is poor. The ataxy of the arms resembles that of Friedreich's disease. The left leg and the left arm are the weaker, and the head is usually slightly drawn down towards the left. The knee-jerks are brisk, L. > R., with slight

left ankle clonus. Plantar responses are flexor. The eye movements and pupils are normal; no nystagmus. Visual fields and acuity normal. Discs normal.

The case appears to be a combination of spastic paralysis and chronic chorea, and might be labelled "spastic chorea." The history of onset and absence of heredity seem to exclude Huntingdon's chorea.

DR. A. F. VOELCKER.—*A case of acute ataxy (encephalitis cerebelli).*

[5] D. F., girl, aged 4½ years, was admitted to the Children's Hospital "for weakness and paralysis" on March 7, 1905.

On December 24, 1904, a rash like that of scarlet fever was noticed all over the body. The child became feverish and listless, and on the fifth day was in great pain, both legs were drawn up and the child was a little rigid. She then became unconscious for fourteen days, during which time there were occasional very slight twitchings of the face, and she occasionally put her hand up to the right side of the head. After the third week of illness she recovered consciousness, but has been unable to stand, feed herself, or speak properly since.

There has been neither otorrhœa nor vomiting.

The mother had chorea as a child, but beyond this the family history is unimportant.

State on admission.—Well grown, rather heavy aspect, lies down in bed and cannot sit up alone. She understands what is said to her. Speech very slow, no stammering or elision. Says very few things beyond her name, address, "yes" and "no." When sat up there is tremor of the head, which tends to fall forward. Cannot stand alone, but with support tends to exhibit some cross-legged progression. The feet are extended. No rigidity of limbs. Marked ataxy of both upper and lower limbs. Irregular choreiform movements of the hands. Marked intention tremor.

Cranial nerves.—No optic neuritis. No nystagmus. No squint or ocular palsy. Pupils equal and react to light and accommodation. No facial palsy. Hears well. Tongue protruded a little to the right.

Reflexes.—Plantar variable. Knee-jerks brisk. No ankle-clonus.

Sphincters.—No loss of control.

No evidence of disease in thorax or abdomen. Temperature 97 to 100°.

Progress.—Slight improvement in speech since admission.

Dr. T. D. Savill.—*Two cases of generalised tonic muscular spasm in middle-aged men, probably paralysis agitans.*

[6] (*a*) O. J. C., a schoolmaster, aged 38. The present illness began when he was 36, in the summer of 1902, with stiffness, slight shaking, and sometimes pain (chiefly in the arms), which was followed in the winter, 1902-1903, by stiffness and dragging of the legs. But he was able to play tennis and bicycle up to the summer of 1903, and he kept at work until Christmas, 1903.

When he first came under notice in June, 1904, there was a generalised stiffness of all the muscles, including those of the face and chest. Every movement was slow and deliberate, both feet were scraped along the ground, especially the left. The gaze and attitude were fixed, and he bent forward during progression. Expression mask-like. Small tremor of the hands, even when supported. Grip of hands about $\frac{5}{6}$ths of normal; muscular power good, but could not walk more than half a mile without increased stiffness and fatigue. Pupils normal, ocular movements stiff; no affection of cranial nerves. No sensory troubles. Skin of face glossy and chloasma across forehead. No history of syphilis.

The symptoms at the present time (March, 1905) remain much the same, but his power of moving his limbs has improved considerably under (1) galvanism to the muscles of the limbs for two months, and (2) high-frequency electricity—auto-condensation couch, effleuve and sparking to muscles. He can now walk two miles without undue fatigue.

[7] (*b*) H. F., a bricklayer, aged 44, came to the West End Hospital March 7, 1905, complaining of *stiffness* and loss of power in the arms and legs, which had started gradually eighteen months before. He said that the hands were first affected so that he could not hold the bricks, and soon afterwards the legs became involved. Nine months ago he first noticed a difficulty in speaking when he was excited.

Now there is a generalised muscular stiffness and slowness of movement. There is an uncertainty in the movements rather than an actual tremor. The gaze and attitude are fixed. The gait is stiff, and he has unsteadiness in turning, but there is no Rombergism or definite inco-ordination. Patellar reflexes increased, no Babinski's sign. The grasp is somewhat deficient, but muscular power everywhere is fair. The speech is deliberate and slightly explosive. The pupils react normally, and there is no ptosis, the fixity of the face suggests a rigidity of facial muscles. No obvious affection of the cranial nerves. There is no definite history of syphilis, but four of his sisters and one brother

have died of consumption. The muscular power soon becomes exhausted, and the question of myasthenia gravis was considered, but there is no faradic reaction of exhaustion.

Dr. LEONARD GUTHRIE.—*Primary atrophy of optic nerves, with partial ophthalmoplegia and cerebellar gait.*

[8] W. L., aged 52, cabman, has complained of difficulty in walking for five or six years. Two and a half years ago he was seized whilst driving his cab by giddiness and temporary diplopia. Two years ago, whilst in bed, his left leg "kicked out" involuntarily, and he felt sensation of "pins and needles" in the fingers of his left hand. He did not lose consciousness. He has since complained of increasing weakness of legs, especially left, and unsteadiness and difficulty in walking.

Present condition.—Healthy-looking, ruddy-faced man. Heart, vessels, lungs and urine are normal. Pupils equal, reactions normal. There is some limitation of upward movements of eyes, and deficient convergence, with slow nystagmoid twitchings on external deviation. The optic discs are very pale, slightly excavated, the cribriform plates exposed, and vessels diminished in size. There is a ring of choroidal atrophy round the right disc. Vision is fairly good. The auditory and the other cranial nerves are unaffected. He has never suffered from headache or vomiting. Mind and intellect are clear. Tremors, ataxy, and pains are absent, sensation is normal everywhere. There is some weakness of the left hand-grasp. The knee-jerks are exaggerated, ankle-clonus is absent, plantar response is negative. Romberg's sign present. Gait is extremely unsteady. He reels and lurches from side to side with special tendency to veer towards the left. He drags his left foot, and dares not walk without support lest he should fall. The lesion is probably of the corpora quadrigemina.

Dr. HARRY CAMPBELL.

[9] (*a*) W. J. L., aged 73, bricklayer. Eighteen years ago tremor in thumb of left hand. Gradually increased, and about ten years ago tremor began in right hand, and interfered with writing. In all other respects seems to be healthy, and can walk long distances without fatigue. Knee-jerks active.

This patient is shown on account of the fact that his daughter (who is also shown to-night) has disseminated sclerosis.

[10] (*b*) J. A., aged 39. Four years ago began to see double. Three and a half years ago walking became difficult, and shortly after, the right hand began to tremble. Six months ago diplopia disappeared.

Present condition.—Pupils unequal. No nystagmus. Well-marked intention tremors especially right side. Knee-jerks greatly increased, ankle-clonus and Babinski's sign present. Speech, which was once very fluent, somewhat syllabic.

This patient is shown because her father (who is shown to-night) has had intention tremor for eighteen years.

DR. HEAD.—*A case of Huntingdon's Chorea.*

[11] H. S., aged 38. A little more than twelve months ago he noticed that his legs began to tremble. About seven months ago his speech became affected. He is married and has four healthy children. His brothers and sisters and their children are healthy. No similar condition has appeared in any of his relatives except in his mother's sister who is now 83 years of age. At the age of 40 she is said to have begun to be restless, as he is now, and to have remained so until the present time.

He is a well-developed man, with no disease in the heart or lungs; the urine is unaffected. He speaks like a man who has difficulty in controlling his tongue; each word is begun with some difficulty, and some words are slurred, especially when he is in the presence of strangers. The tongue is extremely unsteady. Face, hands, and legs are in constant restless movement, which is said to cease during sleep. Chest wall and diaphragm participate in these irregular movements. Tremor is absent. The knee-jerks are normal, and the toes go downwards when the sole of the foot is stimulated. The pupils act normally, and micturition is not affected.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Society can obtain the Index of "Brain" for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 8s. 6d., post free.

To those who are not members of the Neurological Society the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.